"A PROSPECTIVE CLINICAL STUDY OF ARRHYTHMIAS IN ACUTE MYOCARDIAL INFARCTION"

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This is to certify that this dissertation entitled **"A prospective clinical study of arrhythmias in acute myocardial infarction"** is the bonafide work of **Dr P.Vijayaraja.,** in partial fulfillment of the university regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai, for **M.D General Medicine Branch I** examination to be held in **April 2015.**

Captain Dr.B. SANTHAKUMAR , M.Sc(F.Sc) , M.D(F.M)., PGDMLE.,

Dip.N.B (F.M) .,

THE DEAN

Madurai Medical College and Government Rajaji Hospital,

Madurai

Certificate from the HOD

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Dr. S. VadivelMurugan, M.D.

Professor and HOD,

Department Of General Medicine,

Government Rajaji Hospital,

Madurai Medical College,

Madura**i**

Certificate from the GUIDE

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Dr. S.Vadivel murugan,

Professor and HOD ,

Department Of General Medicine,

Government Rajaji Hospital,

Madurai Medical College,

Madurai.

DECLARATION

I , DR P.VIJAYARAJA , solemnly declare that this dissertation titled **"A prospective clinical study of arrhythmias in acute myocardial infarction "** is a bonafide record of work done by at the Department Of General Medicine , Government Rajaji Hospital , Madurai , under the guidance of

Dr. S.VADIVEL MURUGAN. ,M.D, Professor , Department of General Medicine , Madurai Medical college , Madurai.

This dissertation is submitted to The Tamil Nadu Dr. M.G.R Medical University, Chennai in partial fulfillment of the rules and regulations for the award of **M.D Degree General Medicine Branch- I**; examination to be held in **April 2015.**

Place: Madurai

Date:

Dr.P.Vijayaraja.

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CONTENTS

ABSTRACT

A PROSPECTIVE CLINICAL STUDY OF ARRHYTHMIAS IN ACUTE MYOCARDIAL INFARCTION.

AIMS AND OBJECTIVES :

To study the incidence of arrhythmias in Acute Myocardial Infarction (AMI) with respect to type of arrhythmia, age distribution, sex and location of infarction.

MATERIALS AND METHODS :

STUDY POPULATION:

This study is to be conducted among 100 cases of acute myocardial infarction admitted in department of medicine and department of cardiology.

INCLUSION CRITERIA

- Patients admitted with hyper-acute to acute phase of myocardial infarctio

EXCLUSION CRITERIA

1. The patients with previous history of myocardial infarction.
2. A known case of valvular heart disease.
3. A known case of thyroid disorders.

STUDY PROTOCOL:

1. Patients who are admitted with acute myocardial infarction included in the study.

2. Blood investigations , , ecg ,echocardiography are obtained during admission.

3. ECG is recorded at admission at 24 hours ,48 hours and at the time of arrhythmia.The pattern of arrhythmia is noted by using multi parameter monitor.After 48 hours ,all the patients are regularly monitered by 12 lead ecg thrice a day until discharge from hospital .

Results and interpretation :

A total of 100 patients are recruited on admission to the intensive coronary care unit at Government Rajaji Hospital. They included 74 males and 26 females. Patients with confirmed diagnosis of acute myocardial infarction and satisfying the inclusion and exclusion criteria are included in the study group.

This study showed myocardial infarction was more common among elderly .

In this study myocardial infarction was common among males than females.

In this present study Out of 100 patients , 70 patients had arrhythmias.

In this present study Arrhythmia occurred in 77% of females and in 67.5% of males.

In this present study anterior wall MI occured in 45% of patients ,30% of patients had inferior wall MI ,15% patients had inferior wall and right ventricular MI ,6% of patients had infero lateral MI ,4% of patients had lateral wall MI .

In this present study 38% of patients were hypertensive and 62% of patients were non hypertensives.

In this present study 40% of patients were diabetics and 60% of patients were non diabetics.

In this present study 55%% of patients were smokers and 45% of patients were non smokers .

In this present study 65%% of patients were alcoholics and 35% of patients were non alcoholics .

In this present study 62 % of patients were thrombolysed and 38% of patients were not thrombolysed .

In this present study 70 % of arrhythmia occured during the 1st hour ,24% of patients had arrhythmias during 1 to 12 hours ,3 % of patients had arrhythmias during 12 to 24 hours .

82% of smokers had arrhythmia, it was statistically significant.

70% of those consuming alcohol had arrhythmia and was statistical significant.

Arrhythmia occurred more in those who were thrombolysed than in those who were not thrombolysed .It was statistically significant.

86% of of patients with LVEF > 40 % had arrhythmia . It was not statistically significant.

In AWMI majority of patients in whom 2-D ECHO was done,had L.V. dysfunction. In IWMI majority did not have L.V. dysfunction.

Frequency of arrhythmias :

Vpc - 20% , Vt - 5.71%, Vf - 2.85%

Sinus bradycardia - 25.71%

Sinus tachycardia - 2.85%

Atrial fibrillation - 5.71%

Atrial tachycardia - 2.85%

Lahb - 2.85%

Second degree block - 8.57%

Complete heart block - 8.57%

Chb+ 1st degree block - 2.85%

Vpc+ sinus bradycardia - 5.71%

Vpc + sinus tachycardia 2 2.85%

Vpc+ Rbbb 2 2.85%.

SUMMARY

1. The current study in a hospital based descriptive study including 100 patients admitted with acute myocardial infarction.
2. Maximum arrhythmia were observed in the age group ranging from 50 to 60 years.
3. Arrhythmias were more common among elderly male.
4. Ventricular premature contraction was a second most common arrhythmia. However ventricular premature contractions also occurred along with other arrhythmias.
5. Majority of the arrhythmia occurred during the first hour of hospitalization.
6. Majority of the arrhythmias underwent spontaneous resolution.
7. Arrhythmias were noted more in those who underwent thrombolysis.

INTRODUCTION

Coronary artery diseases are the main cause of mortality in developed world even with effective available treatment.

The incidence of, morbidity and mortality are higher among indians than other ethnic groups . The increased mortality in myocardial infarction is due to development of arrhythmias.

Cardiac rhythm disturbances occur in most of the patients during acute myocardial infarction, and approximately twenty-five percent have cardiac conduction disturbance within 24 hours following infarct onset .

Almost any rhythm disturbance can be associated with acute myocardial infarction, including bradyarrhythmias, supraventricular tachyarrhythmias, ventricular arrhythmias, and atrio ventricular block.

With the advent of thrombolytic therapy,coronary reperfusion also produce some rhythm disturbances in patients with acute myocardial infarction .

The purpose of this study is to evaluate the incidence and profile of cardiac arrhythmias in relation to age ,sex , location of infarction in acute myocardial infarction most of the arrhythmias occur in the first 48 hours of hospitalization.

AIMS AND OBJECTIVES

The aim of the present study is to study the clinical profile of arrhythmias in acute myocardial infarction during the first week of admission.

REVIEW OF LITERATURE

HISTORICAL ASPECTS

It is perhaps impossible to identify the very first person who observed variations in the cardiac rhythm.

However, the review of history of medicine, in this regard is helpful in identifying at least some milestones in our understanding of this clinical problem.

Among the ancient times, it is said that Egyptians were aware of the importance of the examination of pulse as early as 13t h Century before Christ.

Chinese considered it as a key to diagnose many condition in 6th century B.C. Just around this time, it is said that 600 types of pulses were known to Ayurvedic physicians.

In the year 1628 A.D, Sir William Harvey described circulation of blood. In the year 1776, William Withering recognized the irregular pulse of atrial fibrillation.

In 1835, Boulland recognised two important abnormalities in the pulse, which he called pulse intermittens and ataxia of the pulse (possibly atrial fibrillation).

Nothangel likened the irregularity of the heart rhythm in ataxia of the pulse to the delirious state of the brain and hence called it ‘celirium cordis’.

In 1855, Nothangel produced kymographic recordings of the irregular contractions .

The description of the irregularity of the pulse in atrial fibrillation:In this form of arrhythmia, there is total irregularity of pulse. At the same time, the height of the individual pulse waves are continually changing’.

Some of the eminent personalities who have contributed to our understanding of this clinical problem are as follows:

Ludwig Aschoff (1866 - 1942) described the conduction system in mammalian hearts and later in 1937 published observations on the conduction system in the congenital anomalies of the heart.

Surao Tawara (1873 - 1952) described the A.V. node. Purkinje described the existence of intraventricular conducting system (1839).

Arthur Keith and Martin W. Flock confirmed Tawaras findings. They also identified the S.A. node.

James Mackenzie (1853 - 1925) was an outstanding physician who studied cardiac dysrhythmias methodically.

William His (1863 - 1964) described the bundle of His.

Walnig in 1926, Kael F. Wenckeback described the intermodal connections.

Backmann in 1916 described the Backmann's intermodal connection.

Electrocardiograph was the first diagnostic tool for understanding dysrhythmias was the product of contributions of many great men like Gabriel Lipman, Augustus Disire Waller, Einthoven and many others. the heart.

Mobitz in 1924 explained Mobitz II degree Heart block.

Frank Wilson and Baker in 1934 described Right Bundle Branchblock.

Grant in 1956 and 1959 introduced the concept of peri-infarction block.Based on various studies Madsen (1983) developed a scoring system for the prognosis in myocardial infarction, which includes heart failure, ventricular tachycardia, atrioventricular block, previous infarction or extension of infarction.

Early deaths are not related to the severity of infarct but observations from monitoring units suggest that the mechanism in most of the cases is arrhythmias and cardiac asystole.

In the recent years the treatment for acute cardiac emergency condition has been dramatically improved due to better understanding of diseases and improvement in diagnostic aspects . Electrocardiograph was the first diagnostic tool for understanding dysrhythmias was the product of contributions of many great men like Gabriel Lipman, Augustus Disire Waller, Einthoven and many others.

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Based on various studies Madsen (1983) developed a scoring system for the prognosis in myocardial infarction, which includes heart failure, ventricular tachycardia, atrioventricular block, previous infarction or extension of infarction.

Other findings that augue poorly are repetitive ventricular ectopic activity, , Q waves in multiple leads persistent horizontal or downsloping ST segment depression, atrial fibrillation, and voltage criteria for left ventricular hypertrophy, an abnormal signal averaged ECG, left ventricular dysfunction.

Deaths most commonly occur within one hour of acute myocardial infarctions.

Early deaths are not related to the severity of infarct but observations from monitoring units suggest that the mechanism in most of the cases is arrhythmias and cardiac asystole.

In recent years the use of cardioversion, artificial pacemaker, anti arrhythmic drugs and particularly thrombolytic therapy has dramatically improved the immediate appropriate care in acute cardiac emergencies.

Our knowledge on the natural history , the prognosis of AMI, and incidence ofarrhythmias in hospitalized patients have dramatically improved due to intensive coronary care monitoring.

Newer investigation modalities like Thallium-201, Pottasium-43, Rubidium- 81 scintigraphy have definite advantage over conventional exercise ECG in demonstrating the site and nature of ischaemia and in analyzing the genesis of complete heart block .

THE ANATOMY AND HAEMODYNAMICS OF CARDIAC DYSRHYTHMIA

It is essential to have a thorough knowledge of the anatomy of the human heart in order to understand the fundamental mechanisms responsible for various cardiac dysrhythmias.

Anatomical structures pertinent in relation to cardiac dysrhythmias include the various cardiac pacemaker and conduct ion systems.

The major topics discussed are Sino – atrial node, Atrio – ventricular node, Common bundle, Bundle ranches and Purkinje fibres. Accessory fibres, such as bundle of Kent, Mahaim's fibres, internodal tracts are briefly discussed.

SINUS NODE: The S - A node is the pacemaker of the heart and it has the following features:

a) SITE: One millimeter below the epicardium of the sulcus terminalis in the right atrium

b) MORPHOLOGY: The S - A node s a crescent structure, having a 'head', 'body', and 'tail', and backwards to the left downwards curling so - to speak around the superior vena cava.

DIMENSIONS:

Length: 15 mm (head, body and tail)

Width: 5 mm (from superior venacava to atrial margins)

Thickness: 2 mm (from epicardial to endocardial surfaces).

CELLULAR STRUCTURE:

The cell types in the S - A node:

1. Nodal cells

2. Transition cells

3.Atrial muscle cells

Nodal cells :

The source of initiation of normal impulse in the node(pace maker cells). They are, primitive cell small, (5 – 10 micrometer) ovoid ,with few organelles and myofibrils in the cytoplasm.

TRANSITIONAL CELLS:

The sinus impulse spread from nodal cells to rest of the myocardium occurs via transitional cells .

INNERVATION:

Both adrenergic and cholinergic nerve endings supplies the sinus node .The automaticity and conduction of the sinus node is increased by adrenergic stimulation and decreased by increased vagal tone.

THE PREFERENTIAL INTER-NODAL AND INTRAATRIAL PATHWAY

There are three such preferential pathways between the sino atrial and Atrio ventricular node.

1) The anterior inter nodal tract - this has two branches - Bachman's bundle and descending branch.

2) The middle inter nodal tract (Wenkebach bundle)

3) The posterior inter nodal tract (Thorel's pathway).

CONDUCTING SYSTEM OF THE HEART:

1. THE ANTERIOR INTERNODAL TRACT:

The anterior inter - nodal tract divides into two branches.

1. The inter atrial branch (Bachman's bundle) - from the right atrium to the left atrium.

2. The descending branch - from the inter atrial septum to the crest of the A - V node.

2. THE MIDDLE INTERNODAL TRACT (WENKIBACH'S BUNDLE):

The middle internodal tract descends in the interatrial

septum - anterior to the fossa ovalis and merges with the fibres of the anterior

internodal tracts to enter the superior crest of the A –V node.

3. POSTERIOR INTERNODAL TRACT (THOREL'S PATHWAYS):

The posterior internodal tract enter the posterior margin of the A - V node. This tract is longer than the other two tracts.

THE BYPASS TRACT:

The bypass tract is a short tract which is mainly a continuation of the posterior internodal tract, but also receives fibres from the anterior and middle internodal tracts. This tract is so named because it bypasses the main body of A - V node to enter it distally. It may also enter the bundle of His directly. Fibres from all three intermodal tracts bend or intermingle just proximal to the A - V node where all three tracts divide into two divisions, one entering the crest of the A–V node, and the other contributing to the bypass tract.

THE ATRIO - VENTRICULAR NODE:

The AV node is a oblong flattened structure convex on one side and concave on the other. The AV node lies on the right side of the central fibrous body - the body.

The anterior or distal end blends imperceptibly with the bundle of His. The AV nodal artery supplies the AV node.

The AV node is connected with the atrial myocardium through the anterior, middle and posterior internodal tracts. It is continued distally as the bundle of His.

In addition, specialized fibres known a para specific fibres of Mahaim may leave the AV node and end blindly in the interventricular septum.

CHARACTERISTIC OF AV NODE CELLS:

- It is the specialized myocardial cells for slow conduction.
- Contain large number of small cells.
- Depolarisation waves pass perpendicular toAV nonaal cells.
- Gap junction between the cells are very minimal.
- Calcium influx is slow.
- Resting membrane potential is (-60 mv)

THE BUNDLE OF HIS - THE COMMON AV NODE:

The distal end of the AV node continues as the bundle of His. The external morphology does not reveal a sharp demarcation between the two, The internal structure of the AV node has a labyrinthine structure of the interweaving strands of cells.

The AV node and bundle of His are sometimes collectively referred to as 'AV JUNCTION'. Electrophysiological The AV node is divided An, N and NH regions.

CHARACTERISTIC OF PURKINJI FIBRES:

- Specialised fast conducting myocardial cells.
- Depolarisation waves pass to the same direction of cells.
- Cells are large in size so there is no much of resistance for conduction.
- More gap junction .
- Sodium entry is very fast.
- Resting membrane potential is -90mv.

THE RIGHT BUNDLE BRANCH: (RBB)

The right bundle branch is situated beneath the epicardium. It runs along the right side of the interventricular septum to the base of the anterior papillary muscle.

It gives of relatively few branches until it reaches the anterior papillary muscle where it begins to ramify breaking up into a network of small branches.

Bundle branch block is primarily an electrocardiographic diagnosis arising from situations that delay or interrupt the passage of an electrical impulse into or within, the ventricles.

Although there is some evidence that BBB can be produced by focal lesions within the bundle of His, most BBB is due to lesions below the bundle of His.

BBB should not be confused with AV block, a condition that implicates delayed or blocked conduction from the atria in or around the AV node into the ventricles.

The bundle branch blocks may occur in the right or left bundles and may be complete or incomplete.

The anterior and posterior fascicles of the left bundle may be blocked, causing characteristic conduction abnormalities. And Fascicular blocks may occur independently or in conjunction with RBBB. Conduction abnormalities can occur in areas surrounding MI. These were formerly termed peri-infarction blocks.

Conduction abnormalities may also result from obstruction in the Purkinje system. These are termed as parietal blocks.

Fascicular blocks do not cause a very significant prolongation of QRS duration, but bundle branch, infarction and parietal blocks are all associated with definite prolongation of QS duration (intra – ventricular condition time).

The intraventricular conduction time can also be prolonged by WPW syndrome, ventricular hypertrophy, aberrant conduction, hyperkalaemia,

certain anti dysrhythmic drugs and in the ventricular or electronically paced rhythm.

THE LEFT BUNDLE BRANCH :

The LBB runs along the left side of the interventricular septum and emerges below the posterior aortic valve cusp.

a) **The antero - superior division:**

This is the most important division which supplies the antero – superior aspect of the LV - the greater part of LV. Delay or block within this division will result in a posterior hemiblock**.**

b) The posterior - inferior division:

A division which supplies the postero – inferior aspect of the LV. Delay or block within this division will result in a posterior hemiblock. Recent studies by Kulbertus and Demonllin revealed a third division of LBB called centro – septal branch.

It probably plays an important role in phasic aberrant ventricular conduction and established intraventricular conduction effect.

THE PARASPECIFIC FIBRES OF MAHAIM

These are muscular conduction fibres which may emanate from the AV node, the bundle of His and the main bundle end blindly, in the septal myocardium.

These fibres could conceivably enable the activation front to bypass the main bundle of His and BB, and thereby activate ventricular myocardium directly.

The fibres may also form the anatomical substances for those of the WPW syndrome associated with the normal prolonged P - R interval**.**

VASCULAR SUPPLY:

THE CORONARY ARTERIES:

The ostia of the two coronary arteries are located behind the aortic cusps near the top of the sinuses of valsalva.

These cusps are designated the right or left coronary cusp, depending on the coronary artery originating from it. The remaining coronary cusps is the non - coronary (or posterior) cusp.

The left coronary artery is ostium posterior and superior to the right coronary ostium due to rotation and silght tilting of aortic root.

LEFT CORONARY ARTERY:

The left main coronary artery (LMCA) travels anteriorly slightly inferiorly and leftward from the left aortic sinus to emerge from behind the pulmonary trunk.

BRANCHES:

- Left anterior descending coronary artery,
- Left circumflex coronary artery
- Diagonal (or intermediate) coronary artery.

Left anterior descending coronary artery:

The direct continuation of left main stem artery is the left anterior descending coronary artery coursing anteriorly and caudally over the ventricular septum within the anterior interventricular sulcus. The branches of this artery, in their usual order of origin, are the first diagonal, the first septal perforator, right ventricular branches (not always seen in normal hearts), other septal perforators and other diagonal branches.

There may be 2 - 6 diagonal arteries, including the first diagonal, which may originate separately from the left main trunk. The free wall of the left ventricle is supplied by diagonal branches.

RIGHT CORONARY ARTERY:

From the right aortic sinus , right coronary artery leaves and descends curving posteriorly in the right atrioventricular groove.

The branches of the right coronary artery include the conus artery to the right ventricular outflow area, the artery to the sinusnode , right atrial branches, the acute marginal branches, several anterior RV branches ,the artery to the AV node and proximal bundle branches, the posterior descending artery, and terminal branches to the LV and LA.

When the sinus node artery originate from the right coronary artery, it runs along the anterior right atrium to the superior venacava, which it encircles in a clockwise direction before it penetrates the sinus node.

In 40 - 50 percent of the hearts, the sinus node artery originate from the proximal left circumflex artery and crosses behind the artery and in front of the LA to reach the superior venacava. Along its course, the sinus node arteries supply the atrial myocardium, the crista terminalis and the atrial septum.

The artery to the AV node arises at or near the inward U - shaped turn of the posterior descending coronary at the level of the crux of the heart.

Usually, the posterior descending artery descends proximal to the crux and courses towards the apex within the posterior interventricular septum or sulcus.

The right coronary artery provides blood flow to the sinus node (50 - 60 percent), the RV wall and the right atrium. In 85 - 90 percent of the hearts, the posterior branch supplies the basal one half to the two - thirds of the posterior ventricular septum, the proximal bundle of His , AV node, half the diaphragmatic surface and the posteromedial papillary muscle of the LV.

Through communicating vessels the conus coronary artery makes collaterals between right coronary and the anterior descending coronary

artery. Kugel's artery makes collateral between the AV node and the posterior - circulation.

THE AUTONOMIC NERVE SUPPLY OF THE HEART:

The heart is supplied by both parasympathetic and sympathetic fibres. Parasympathetic innervation consists of branches from right and left vagi.

The SA node is supplied by right vagus and the AV node is supplied by the left vagus .The sympathetic system innervates both atria and ventricles.

ELECTROPHYSIOLOGY OF THE HEART

The knowledge of electrophysiology of cardiac excitation has been greatly extended by direct intracellular recording. A microelectrode inside a single myocardial fibre paired with a extracellular electrode enables to measure the electrical potential inside and outside the muscle fibre and changes that occur during the activity to be recorded (Hoffman and Cranefield 1960).

The difference in the potential is termed 'transmembrane potential', and the changes during the activity are termed 'action current. During the resting phase, when the fibre is said to be polarized, the inside is electrically negative compared with its outside.

The resting transmembrane potential is surprisingly large, measuring 90mv. During the first phase of action current, the transmembrane potential falls rapidly to zero and transiently overshoots (phase 2), so that the inside of the fibre becomes approximately 30 MV positive to the outside.

This transient overshoot is followed by a plateau (phase 3) and then the more gradual downstroke of repolarization (phase 4) follows. The ionic concentration of the intracellular fluid of the cardiac muscle fibres like that of all living cells is markedly different from that of extracellular fluid. Since the cell membrane is ordinarily permeable to all simple ions it is necessary to explain how the ionic composition of the intracellular fluid is maintained.

The generally accepted view is that the cells contain 'metabolic sodium pumps', which continuously eject sodium ions from the cell. Since this is achieved in the phase of both electrical and chemical gradients, active metabolic work is involved. The electrical changes represented by the action current are brought about by rapid fluxes of ions across the cell membrane.

It is believed that both depolarization and repolarization are passive events resulting from abrupt changes in permeability of cell membrane to sodium ions. Sodium ions then rapidly enter the cell as a result of electrochemical gradient and the upstroke of the action current is recorded.

Repolarization is initially achieved by an increase in potassium permeability so that rapid egress of positively charged potassium ions from the

cell occurs and the transmembrane potential is restored. This Regress of potassium ions is facilitated by the large chemical gradient and the disappearance of electrical gradient following depolarization. At the end of the action current, the inside fibres will have gained sodium ions and lost potassium ions. Following activity the original differential ionic concentration are restored by the metabolic pumps.

The action currents of atrial and ventricular muscle fibres is that the transmembrane potential remains constant during diastole.On the other hand, the action current of pacemaker cells is characterized by slow spontaneous depolarization during diastole. For reasons not clear, once the transmembrane potential has fallen tothreshold value, which is approximately 40 mv, rapid depolarization follows and this automatically fires off a propagated response which will be conducted over the heart.

The AV node is activated early during the P - wave o f surface ECG. The main delay in AV transmission occurs at the atrial margins of the AV node where the conduction velocity is a very low 50mm per second. Within the node, conduction velocity progressively increases to reach 1000–1500 mm per sec. During the retrograde conduction from ventricles to atria, the same conduction velocities occur except at the atrial margin of the node where retrograde conduction is even slower than anterograde.

The development of intracardiac electrocardiographic recording and stimulation techniques has brought about a renaissance in cardiac electrophysiology. It simplifies the electrical activity that is inconspicuous and unrecordable in the surface ECGs. Local ECGs may be recorded in the intact human heart from the atrial tissue adjacent to the SA node, low intraatrial septum, coronary sinus, bundle of His, right and left bundle branches.

Amplifications of atrial activity that is inapparent in the surface ECG has been accomplished by intraoesophageal lead and intraatrial electrogram. Recording of the AV conduction system was first discovered by Scherlong and his colleagues 1969.

HAEMODYNAMIC CONSEQUENCES OF DYSRHYTHMIA;

The haemodynamic consequences of cardiac dysrhythmia vary with and are dependent upon the heart rate, the regularity of the cardiac rhythm, the relationship between atrial ventricular systoles, the synchronization of ventricular contraction, the presence or absence and severity and nature of underlying heart disease, the presence or absence of drug toxicity (particularly digitalis) and the status of vasomotor control mechanism.

FACTORS AFFECTING HAEMODYNAMIC CONSEQUENCES OF CARDIAC DYSRHYTHMIAS:

1. Heart rate.

2. Irregularity of rhythm.

3. Relationship between atrial and ventricular contraction.

4. Synchronization of ventricular contraction.

5 Cardiac disease.

6 Influence of drugs.

7 Vasomotor control mechanisms

During cardiac dysrhythmias significant circulatory impairment has been documented in various organs. The main burden will be on coronary circulation, cerebral circulation, renal circulation mesenteric circulation and musculocutaneous circulations in decreasing order.

MECHANISMS RESPONSIBLE FOR CARDIAC DYSRHYTHMIAS:

Dysrhythmias are categorized as due to abnormal impulse formation abnormal impulse propagation, and combined abnormalities of impulse formation and propagation.

Abnormalities of impulse formation include abnormalities of automaticity and early or delayed after depolarizations with triggered activity. Abnormalities of impulse propagation include conduction block and reentry of the cardiac impulse. Combination of abnormalities of both impulse format ion and propagation result in complex dysrhythmias. In addition to general mechanism of dysrhythmogenesis it is useful to know which cardiac tissues participate in the dysrhythmia and the ionic mechanisms and structural abnormalities that promote the dysrhythmia.

The mechanisms responsible for cardiac dysrhythmias are presented below:

Abnormalities of impulse generation:

- Alteration of normal automaticity,
- Delayed after depolarizations.

Abnormalities of impulse conduction:

- ❖ Slowing of conduction and block
- ❖ Unidirectional block and reentry
- ❖ Random reentry
- ❖ Ordered reentry
- ❖ Combined abnormalities of impulse formation and propagation:
- ❖ Conduction slowed by Phase - 4 depolarization Parasystole.

ALTERED NORMAL AUTOMATICITY:

Alteration of the normal automatic mechanism can produce conduction abnormalities. Under the influence of sympathetic activity the SA node fires excessively and failed in diseases condition. Pacemaker in the His Purkinje system may control cardiac rhythm as escape pacemaker. The first situation occurs when sinus bradycardia of AV block reduces propagation of SVT impulses to a rate lower than the intrinsic rate of His - Purkinje pacemakers. Also, abnormal conditions, eg., ischaemia or drug treatment (sympathomimetic drugs or phosphodiesterase inhibitors), can lead to an increase in the rate of firing pacemakers in the His - Purkinje system.

ABNORMAL AUTOMATICITY:

Automaticity is the tendency of the cell to undergo spontaneous depolarization. Parasymphathetic stimulation causes deloading of cations in SAnode produces bradycardia,and symphathetic stimulation produces tachycardia by loading of cations.

AFTER DEPOLARIZATIONS AND TRIGGERED ACTIVITY;

Secondary depolarizations that occur after starting repolarizations are called after depolarizations. Triggered activity is single or repetitive firing of a group of cells initiated by an after depolarization. Triggered activity is not a form of automaticity; triggered activity itself is not self – excitatory but rather

depends on a preceding action potential and after depolarizations to initiate a process. Triggered activity can be initiated by early and delayed after depolarization.

EARLY AFTER DEPOLARIZATIONS:

Early after depolarizations are depolarizations that occur before the cell fully repolarizes. They either arise from the plateau of the action potential or during the rapid phase of repolarization. It can occur in any type of heart cell. The conditions that delays the repolarizations of heart cells, eg., hypokalaemia, slow heart rate and drug toxicity commonly produce early after repolarizations. Early after depolarizations can trigger sustained rapid firing in Purkinje fibres.

The torsades de pointes form of VT is very common clinically, initiated by early after depolarizations. when patients have the same condition that are known to produce early after depolarizations experimentally - hypokalaemia, , or long QT, slow heart rate . All treatment approaches has the concept , to increase K + conductance in the heart muscle thereby shorten action potential duration , increasing extracellular K + drugs, increase K + conductance and rapid pacing are beneficial for early after depolarizations caused by decreased extracellular K + concentration.

DELAYED AFTER DEPOLARIZATION:

In thiscondition depolarization occurs after the cell has repolarised. As stimulation becomes more premature ,or the heart rate increases the delayed after depolarization becomes larger and larger ; threshold voltage is reached, a rapid firing or run or triggered activity may be provoked. Increased catecholamine concentrations , rapid pacing, increased (Ca) ++, premature activation, and digitalis toxicity are known to increase the amplitude of delayed after depolarization . Although triggered activity is not self - initiating, it can be self - perpetuating.In digitalis toxicity delayed after depolarization occurs in cardiac Purkinje fibres,due to an abnormal current called ‘transient inward current’ is an ideal example for triggered activity and delayed after depolarization .

PREMATURE STIMULATION:

The two examples of reentrant dysrhythmias are AV nodal reciprocating tachycardia, and recurrent, sustained ventricular tachycardia. The current view is that both of these dysrhythmias have reentrant mechanism.

REENTRANT DYSRHYTHMIAS:

The ventricular reentry can occur in either the Purkinje network or ventricular muscle alone or the reentrant path may include both of these cell types and their junctions.

ANAMALOUS AV CONNECTIONS:

The most convincing example of a reentrant dysrhythmia in humans is the Av reciprocating tachycardia seen in WPW syndrome. During sinus rhythm, AV conduct ion of sinus impulse in WPW uses both the AV node and to anomalous AV connect ion. The additional AV pathway promotes reentrant SVT. Elaborate clinical electrophysiological mapping studies in the catheterization lab or operation room can determine tachycardia pathway precisely. When the anomalous connect ion is divided, it becomes impossible to initiate tachycardia. This is conclusive evidence for reentry.

EXCITABLE GAP AND ENTRAINMENT OF VENTRICULAR – TACHYCARDIA:

In WPW syndrome most of the tissues in the circuit fully recovered during a cycle of AV reciprocating tachycardia. In order to start the reentrant rhythm one way block is required . Very slow conduction restricted to small part of the circuit is the critical factor in sustaining these reentrant tachycardia. eg. AV node in the case of AV reciprocating tachycardia in the WPW syndrome .A premature impulse timed so that it blocks the tachycardia wave front in the retrograde limb but fails to propagate in the antegrade direction will terminate the tachycardia.

STATIC AND DYNAMIC BLOCK AND REENTRY:

Experimentally, In subacute and chronically infarcted ventricles reentrant ventricular tachycardia can be initiated. The Static barriers created by infarcted tissues, becomes dynamic due to abnormal behaviour of ischaemically damaged but surviving cells in the epicardial rim overlying the infarcted zone. The wave fronts can bock, collide and extinguish, or summate.

REENTRY DUE TO ANISOTROPY

As the infarcts heals, fibrosis separates the surviving muscle bundles is the epicardium over the infarct reducing side - side connection. Measurement s of conduction on the epicardium over healed infarcts show marked an - isotropy of conduction - conduction velocity is about four times as rapid in a direction parallel to the fibre orientation as in the transverse direction. Reentry may occur along the long axis of fiber orientation, and ventricular tachycardia can result.

DYSRHYTHMIA DUE TO CURRENT OF INJURY:

In myocardial infarction a current of injury can flow across boundaries and it can be strong enough to excite adjacent ventricular muscle or Purkinje fibers . This mechanism might operate for brief periods of time after acute infarction.

ARRHYTHMIAS COMPLICATING ACUTE MYOCARDIAL INFARCTION

In acute myocardial infarction arrhythmias occurs either early phase of MI (first 30 minutes after coronary occlusion) or late phases in the infarction process.

Mechanism:

- Micro – reentry.
- Slowing of conduction,
- Alterations in refractoriness and excitability,
- Abnormal automatic impulse formation.
- Loss of trans membrane resting potential,

The concentration of potassium level increased in ischemic zone whereas cells in between normal myocardium and ischemic region are only partially depolarized and therefore have larger amplitude action potentials . Polymorphic VT and ventricular fibrillation occurs in markedly injured myocardium due to slowing of impulse conduction . Reperfusion arrhythmias occur due to washout of potassium , lactate and toxic substances from ischemic zone..

Cells in reperfused myocardial zones can exhibit action potentials of the slow response type. The extent of myocardial infarction determines the

severity of fatal arrhythmias like ventricular fibrillation and the incidence of malignant ventricular arrhythmia.

Hemodynamic consequence

In patients with acute MI cardiac output is depressed by both bradycardia and tachycardia. At more rapid heart rate the myocardial oxygen demand is more and the supply is less due to shortening of diastolic period, that adversely affect ischemic myocardium.

In patients with AMI, the optimal heart rate is usually in the range of 60 to 80 beats/min .A trial contraction contributes about 15-20 percent of left ventricular filling. For left ventricular filling, atrial systole is of greater importance in patients with reduced diastolic left ventricular compliance of any cause (including AMI) . Atrial systole boosts end-diastolic pressure by 29 percent, stroke volume by 35 percent and end-diastolic volume by 15 percent in patients with AMI.

Table 1: Classification of arrhythmias

Category	Arrhythmia
1.electrical instability	Ventricular pre mature beats Ventricular tachycardia Ventricular fibrillation Accelerated idioventricular rhythm Paroxysomal atrioventricular junctional tachycardia
2.pump failure /excessive sympathetic stimulation	Sinus tachycardia Atrial fibrillationand/or atrial flutter Paroxysomal supraventricular tachycardia
3.bradyarrhythmias and conduction disturbances	Sinus bradycardia Junctional escape rhythm Atrioventricular block and intraventricular block

VENTRICULAR ARRHYTHMIAS

Ventricular arrhythmias occurs in AMI is reversible during the acute phase of the MI, electrolyte abnormalities like low potassium and low magnesium level or from reperfusion. The ventricular arrhythmias that occur late in the course of AMI produce severe consequences than early arrhythmias.

Ventricular Premature Beats:

Site of origin - ventricle at sites remote from the Purkinje network.

QRS complex- wide that is typically >140 ms in duration.

Ventricular premature complexes are common in presence of structural heart disease common and increase with age.

Patterns of Ventricular premature complexes

***Bigeminy*-** in which every sinus beat is followed by a VPC .

Trigeminy- in which two sinus beats are followed by a VPC.

Multiformed **VPCs -** have different morphologie*s.*

Pairs **or** ***couplets*** -Two successive VPCs are termed.

VT- when the rate is >100 beats per minute with three or more consecutive VPCs .

Nonsustained VT.-If the repetitive VPCs terminate spontaneously within 30 seconds.

Interpolated VPCs-VPCs that fail to influence the oncoming sinus impulse.

In VPC the QRS pattern does not typically follow right or left bundle branch block pattern.VPCs are most commonly associated with a "fully compensatory pause. The VPC typically does not conduct to the atrium. In evolving myocardial infarction the incidence of ventricular fibrillation reduced by early administration of beta block if vpbs are associated with tachycardia.

An increased risk of SCD occurs with frequent VPCs and nonsustained VT In patients with structural heart disease. Treatment of VPCs by anti arrhythmic drug therapies eliminate the VPCs ,with increased risk life-threatening arrhythmias (drug-induced QT prolongation and TDP). In the post infarction patient frequent ventricular premature beats (more than 10 per hour) are an independent risk factor for subsequent mortality. The suppression of VPBs in AMI does not improve the survival.

Accelerated Idioventricular Rhythm:

Rate >40 beats per minute and <120 beats per minute

Rhythm – ventricular.

Mechanism- abnormal automaticity :

AIVR is frequently present in the setting of acute myocardial infarction (MI), (upto 20%),digoxin intoxication, acute myocarditis, , and postoperative cardiac surgery, cocainein toxication. Sustained forms of AIVR occur particularly in acute MI and postoperatively. Because of the loss of AV synchrony hemodynamic compromise can occur in the setting of sustained AIVR.

Patients with proximal right coronary artery occlusion with RV infarction associated hemodynamic consequences of AIVR are most susceptible to bradyarrhythmias. Atropine or by atrial pacing may be an important treatment consideration. In these patients,to accelerate the atrial rate. AIVR frequently occurs during the first 48 hours anr rarely occur after 2 days . Accelerated idioventricular rhythm is commonly observed shortly after successful reperfusion. As this arrhythmia is self limiting , there is no increased risk of ventricular fibrillation or death, and treatment in generally not required.

Ventricular Tachycardia

Three or more consecutive VPCs with a rate > 100/ minute is VT .

- Non Sustained VT- if the tachycardia terminates within 30 seconds.

- Sustained VT –if the tachycardia lasts for more than 30 seconds or less than 30 seconds with hemodynamic compramises.
- Monomarphic VT- uniform QRS complex.
- Polymorphic VT- multiform QRS complex.

The differentiation of VT from SVT with aberrancy is

- Unually wide QRS complex.
- Ventricular fusion complex.
- Complete AV dissociation.
- Ventriculo atrial conduction with block.
- Sinus captured complex.

VT occurring late in the course of AMI is more common in patients with transmural infarction and left ventricular dysfunction, is likely to be sustained, usually induces marked hemodynamic deterioration, and is associated with both an increased hospital mortality and long – term mortality.

Management:

- Serum potassium level should be maintained above 4.5 meq/l,
- Serum magnesium levelshould be above 2 meq/l ,
- Rapid polymorphic vt - unsynchronized discharge of 200 joules,
- Monomorphic vt - synchronized discharge of 50-100 joules.
- Lidocaine

- Procainamide
- Amiodarone
- Patients with recurrent vt -hypoxia,hypotension acid base or electrolye disturbance should be corrected
- Refractory vt- procedures such as implantation of antitachycardia devices or surgery.
- ventricular fibrillation or VT from nonreversible causes - ICDs or ICDs

Ventricular fibrilation

Ventricular fibrillation occurs in acute mi in three settings.

1. Primary VF.

2. Secondary VF.

3. Late onset VF.

Primary ventricular fibrillation occurs unexpectedly and suddenly ,either the patients have acute left ventricular failure or without any symptoms.The incidence of primary VF declined rapidly for the past few decades. After the onset of symptoms VF occur within 4 hours in 40% of patients , and within 12 hours in 80% of patients. Secondary VF occur in patients with severe left ventricular failure and cardiogenic shock.

Late ventricular fibrillation occur in patients with massive infarct and severe LV failure.

Higher risk for late in - hospital ventricular fibrillation :

- Anterior wall infarction
- Right ventricular infarction who require ventricular pacing
- atrial flutter, or fibrillation early in the clinical course
- patients with persistent sinus tachycardia
- Patients with intra ventricular conduction defects.

In acute AMI with primary VF the in hospital mortality is around 20%. In patients with AMI and secondary VF the mortality is about 40-60% particularly when it is associated with poor LV function , late onset arrhythmia and cardiogenic shock. The in hospital miortality is progressively declined for the past few decades due to

- Wide use of beta blocking drugs
- More effective treatment of ventricular dysfunction
- More effective treatment of electrolyte imbalances
- Admission of lower risk patients to coronary care units.

In patients with primary VF the short term prognosis is good. Patients surviving early in hospital with Ventricular fibrillation

Has no adverse effect on long term survival after hospital discharge.

Management :

The treatment of VF -Un synchronized electrical countershock - 200-300 joules as rapidly as possible.

Failure of electrical countershock to

- recurrent vt or vf,
- electromechanical dissociation
- electrical asystole.

The causes of repeated VF are

- Acidosis
- Digitalis intoxication ,
- Electrolyte abnormalities and
- Prolonged hypoxemia and

The treatment is correction of abnormalities and repeated electrical counter shock.

Treatment of persistment vf is

epinephrine -intravenous route 1mg i.v

intracardiac route (upto 10 ml of 1:10,000 concentration)) to facilitate a further defibrillation attempt.

Prevention of refractory recurrent

episodes -amiadarone(75-150 mg bolus). -

bretylium tosylate 5mg/kg intravenously .

Intracardiac administration of epinephrine or calcium gluconate

May be attempted to promote restoration of an effective heart beat when there is no myocardial rupture.

Prophylaxis :

- Correction of hypokalemia.
- Correction of hypo magnecemia.
- Intravenous beta blockers

BRADYARRHYTHMIAS

Sinus Bradycardia

Sinus bradycardia is more than twice as common in LCX or RCA infarcts as compared to LAD infarcts. The SA node ,AV nodes and atria and richly innervated by the vagus nerve, and stimulation produce both hypotension and bradycardia (the Bezold – Jarisch reflex) . In patients with sinus bradycardia, the acute mortality rate appears to be low due to increased vagal tone that reduces myocardial oxygen demand.

Atrioventricular and Intraventricular Block

In acute AMI conduction block occur at any level of the AV or intraventricular conduction system.

1. Av block – block at the level of AV node and the bundle of His.
2. Bundle branch block -right or left bundle branch block

3.Fascicular block - left anterior or left posterior (fascicular)divisional blocks.

Complete heart block is common in patients with inferion wall MI, and in hospital mortality is high even with thrombolytic theraphy. In inferior MI heart block is frequently as a result of ischemia, with increased vagal tone, consequent block within the atrio ventricular node .This usually results in Mobitz type I second degree AV or block first - degree AV block and unless there is associated hemodynamic instability it requires only simple observation . In patients with proximal RCA occlusion inferior wall MI withRVMI occur where mobitz type 2 block and complete heart block is common and the mortality is very high. Early reperfusion theraphy is indicated to restore the normal AV conduction and reduce the infarct size..

First - degree AV block

First degree AV block commonly occur in patients with anterior wall MI and associated fascicular block.

The site of origin is at the level of AV node.

When first degree block occurs below the level of AV node the chance of complete heart block and ventricular asystole is high.

Generally it does not required any treatment.

Second - degree AV block:

Mobitz Type I OR Wenckebach AV block:

It occurs in up to 10 percent of patients with AMI .

- Usually associated with narrow QRS complexes
- Usually secondary to ischemic injury
- Generally occurs within the AV node
- occurs more commonly in patients with inferior than anterior MI,
- Usually transient and does not persist for more than 72 hours after infarction,
- Rarely progresses to complete AV block.
- May be intermittent,

First - degree and type I second - degree AV blocks are caused by ischemia of the AV node most commonly associated with occlusion of the right coronary artery and are not appears to affect the survival.

Indication of atropine in mobitz type 1 block :

- ventricular rate falls below 50 beats/min or
- heart failure
- associated bundle branch block
- ventricular irritability .

Mobitz type II AV block:

The overall incidence is less than 1 percent.

Type II second – degree block

- Associated with a wide QRS complex
- Usually originates from a lesion in the conduction system below the bundle of His
- Often reflects trifascicular block with impaired conduction distal to the bundle of His
- Almost always associated with anterior rather than inferior infarction.
- Often progresses suddenly to complete AV block

It should be treated with a temporary trans venous or external pacemaker Because of its potential for progression to complete heart block.

Complete (third - degree) AV block:

Complete AV block can occur in patients with either inferior or anterior wall infarction.

The AV conduction system is supplied by

1 .Septal perforating branch from the left anterior descending coronary artery

2 . AV branch of the right coronary artery

The incidence of Complete AV block is higher in patients with right ventricular infarction.

The prognosis depends on and the size of the infarction the anatomical location of the block in the conduction systems. Complete heart block in inferior infarction results from either supra nodal or intra nodal lesion , often progressing from first -degree or type I second- degree block. The escape rhythm is usually stable often junctional and without asystole, narrow QRS complex with a rate exceeding 40 beats/min and the mortality is high in patients with right ventricular MI. In patients with anterior infarction, third -degree AV block often occurs 12 to 24 hours after the onset of infarction, suddenly although it is usually preceded by intra ventricular block and often Mobitz type II AV block,have unstable escape rhythms with rates less than 40 beats /min with wide QRS complexes.

In this group of patients the mortality in is very high about 70 to 80 percent. Indication of Temporary pacing

- Slow ventricular rate .
- Hypotension
- Pump failure
- Ventricular irritability.

Intraventricular Block:

The incidence of intraventricular block in thrombolytic era is about 2-5% . Both the left anterior descending and right coronary arteries supplies the right bundle branch and the left posterior division whereas septal perforators

originating from the left anterior descending coronary arterysupplies the left anterior division .

Isolated fascicular blocks:

In complete AV isolated left anterior divisional block is rare block. A larger infarct is required to block the posterior fascicle and the mortality is high . Both in anterior and posterior fascicular block Complete AV block is not a frequent.

Right bundle branch block:

The incidence of RBBB in acute MI is approximately 2 percent in patients with acute anteroseptal infarction it may may lead to AV block . In patients with anterior MI with congestive heart failure mortality risk is high when it is associated with isolated right bundle branch block.

Bi fascicular block:

Bi fascicular block is the combination of left anterior and posterior divisional blocks or the combination of right bundle branch block with either left anterior or posterior divisional block. The risk of complete AV block is high, if a block occurs in two of the three divisions of the conduction system, Intra ventricular block produces severe pump failure secondary to the extensive myocardial necrosis and the mortality is also high. 2 to 5 percent of patients with AMI develop left bundle branch block.

If latter progresses to Right bundle branch block, the mortality is very high and also more prone for developing ventricular fibrillation. With cardiac failure and massive infarction the mortality is even high in the absence of complete AV block. The conduction defects acquired during the course of infarct in a patient with AMI have higher chances of developing complete heart block rather than those with pre-existing blocks.

Bi divisional block with first degree AV block indicates disease of third division called " Trifascicular block " where 40% progresses to complete heart block.

- The combination of right bundle branch block and left anterior divisional block,
- Any form of trifascicular block,
- Complete heart block.

ASYSTOLE :

There is higher incidence of asystole if it occurs as a terminal complication and lower incidence if it occurs as a primary event or with conduction abnormalities.

The development of bundle-branch block in acute phase of AMI indicates proximal LAD occlusion and requires immediate intervention.

SUPRAVENTRICULAR TACHYARRHYTHMIAS

Supraventricular arrhythmias and heart block are common in patients with AMI . The clinical presentation can range from a benign, asymptomatic ECG finding to catastrophic hemodynamic collapse. Some of these arrhythmias have long term adverse prognostic significance, whereas others do not. The diagnosis and treatment of these dysrhythmias have advanced in the thrombolytic era.

Virtually every known rhythm disturbance has been described in the setting of AMI . 10 to 30 percent incidence of arrhythmia has been shown. This incidence, however, appears to be reduced in patients who are treated with reperfusion therapy. Supraventricular arrhythmias are associated with a variety of predictors of poor outcome in AMI. Transient hypertension or hypotension occurring in sinus tachycardia is associated with augumented sympathetic activity.

Common causes include,

- persistent pain,
- left ventricular failure,
- fever,
- pulmonary embolism,
- pericarditis,

- hypovolemia,
- anxiety,
- the administration of cardioacelerator drugs such as atropine,
- epinephrine or dopamine,
- rarely it occurs in patients with atrial infarction.

Sinus tachycardia is more common in patients with anterior infarction and significant left ventricular dysfunction. Sinus tachycardia worsens myocardial ischaemia due to increased myocardial oxygen consumption and as well as reduced time available for coronary perfusion. Persistent sinus tachycardia is a poor prognostic sign with excess mortality and signifies persistent heart failure.

Approximately 30% of patients with Acute MI develop sinus tachycardia .The prognostic importance of this rhythm depends upon the underlying cause but sinus tachycardia in the setting of MI is generally associated with worse outcome.

In the pre thrombolytic era it was observed that isolated sinus tachycardia occurred in the absence of other obvious causes such as clinically evident heart failure in 16% of 610 consecutive MI admissions. These patients had both a higher in- hospital and longterm mortality rate than patients without this finding. Administration of beta blockers may be helpful when sinus tachycardia is a manifestation of hyperdynamic circulation.

Atrial premature contractions:

Atrial distension which is caused by increase in left ventricular diastolic pressure can cause Atrial premature contractions and the atrial tachyarrhythmias (paroxysmal supraventricular tachycardia ,atrial flutter, and atrial fibrillation) .This increase in left ventricular diastolic pressure can be due to pericarditis with its associated atrial epicarditis, or rarely by ischemic injury to the sinus node and atria . As cardiac output is unaffected in atrial premature beats , mortality rate is low. Even though no specific treatment is needed, these beats may indicate over stimulation of autonomic nervous system or overt or occult heart failure .

Normal sinus rhythm with premature atrial complexes:

Atrial premature complexes occur in at least 50% of all patients with Acute MI . They may be particularly frequent in patients with heart failure, atrial ischemia /infarction and pericarditis.

Paroxysmal supraventricular tachycardia:

Incidence of Paroxysmal supraventricular tachycardia in acute MI patients is less than 30%. As the ventricular rate is rapid, PSVT must be treated aggressively. Sinus rhythm can be restored by carotid sinus stimulation manually. Intravenous metaprolol (5-15mg), verapamil (5-10mg), diltiazem (15-20 mg) can be used in patients with normal LV function. If there is no

hypotension , adenosine can be given.Rapid atrial stimulation using a trans venous intra atrial electrode or direct current counter shock can be given in patients with hypotension or congestive cardiac failure.

Atrial flutter and fibrillation:

The least common major atrial arrhythmia associated with 1% of patients of Acute MI is atrial flutter. Usually the Atrial flutter is transient and in Acute MI , atrial flutter is due to sympathetic stimulation of the atria which is more common in patients with left ventricular failure or pulmonary emboli .Recurring saw tooth flutter waves at a rate of 250 to 300 per minute, especially in the inferior leads (II, III or AVF) or lead VI are seen in ECG.Depending upon the atrial rate and the degree of AV block (typically 2:1; other blocks like 3:1, 4:1 and variableblock), the ventricular response rate can vary from 75 to 175 per minute. Atrial flutter can either revert to normal sinus rhythm spontaneously or degenerate into atrial fibrillation. If there is a constant relationship of the flutter waves to the conducted QRS complexes , S1 will often have a constant intensity.

Atrial flutter with variable conduction causes variation in the intensity of the S1. In patients with Acute MI, atrial fibrillation is common than flutter in 10 to 20 %. Fibrillation is also usually transient or can be seen with LV dysfunction , in patients with pericarditis and ischemic injury to the atria and right ventricular infarction.

Cardiac output reduces due to rapid ventricular rate and inefficient atrial contraction. In patients with Acute anterior wall MI , atrial fibrillation is associated with increased risk of stroke and mortality. Atrial fibrillation is consider as a independent risk factor for poor prognosis as it indicates extensive infarction and severe homodynamic instability.

Atrial fibrillation can cause rapid hemodynamic

instability by three mechanisms :

- Irregular ventricular filling,
- Increased ventricular response rate with decreased diastolic filling time,
- Loss of the atrial component of the cardiac output,

  The ECG findings :
- Rate - 350 to 600 per minute,
- Irregular atrial base line morphology,
- The ventricular response - irregularly irregular,
- Rate of 100 to 160 per minute in AMI.

The physical findings :

- ❖ Irregularly irregular pulse.
- ❖ Absence of a-waves in the jugular venous pulse,
- ❖ Variation in the intensity of first heart sound,
- ❖ Pulse deficit.

Patients with Atrial fibrillation commonly associated with underlying three vessel disease.

Common association :

- ❖ Older patients with larger infarctions,
- ❖ Worse Killip class ,
- ❖ Higher heart rates ,
- ❖ Higher inhospital mortality,
- ❖ Higher risk of stroke .

The onset of atrial fibrillation is usually after 24 hours due to

- Heart failure - most common
- Atrial ischemia,
- Pericarditis.

Management:

Clinical trials have shown a 45% conversion rate for AF and a 60% conversion rate for AFL. Ibutilide is associated with a 4% to 8% risk for Torsades de pointes (TdP), especially in the first 2 to 4 hours after administration of the drugs. Because of this risk, patients must be monitored on telemetry with an external defibrillator immediately available during ibutilide infusion and for at least 4 hours after ibutilide infusion.

The risk for TdP is higher in patients with cardiomyopathy and CHF. Ibutilide is given as an IV bolus, at a dosage of 1 mg (0.01 mg/kg if patient is <60 kg), infused slowly over 10 minutes. Faster administration can promote TdP . The efficacy of antiarrhythmics to achieve pharmacologic conversion drops sharply when AF is >7 days in duration. For shorter-duration AF episodes, dofetilide, sotalol, flecainide, and propafenone have some efficacy, while amiodarone has limited efficacy to achieve pharmacologic cardioversion.

Maintenance of sinus rhythm with antiarrhythmic agents is associated with a small risk for life-threatening proarrhythmia. As a result, antiarrhythmic therapy should be reserved for patients who have highly symptomatic AF in spite of adequate rate control. Class I agents inhibit the fast sodium channel, class II agents are β-adrenergic antagonists, class III agents primarily block potassium channels, and class IV agents are calcium channel antagonists.

Commonly used antiarrhythmic agents, their major route of elimination, and dosing regimen are listed in Table 6. The most effective agents for maintenance of sinus rhythm are flecainide, propafenone, sotalol, dofetilide, and amiodarone. Flecainide and propafenone are class Ic antiarrhythmic drugs that are useful for maintenance of sinus rhythm in patients with structurally normal hearts. In patients with structural heart

disease, class Ic agents are associated with an increased mortality rate and both agents are potent negative inotropes that can provoke or exacerbate heart failure.

Both agents prolong the QRS duration as an early manifestation of toxicity. The toxic drug levels correlate with heart rate due to preferential blockade of active sodium channels. This property is described as use dependence. Exercise ECG can be used to give additional information about dose safety at high heart rates.

Flecainide should be used with caution without concomitant dosing with an AV nodal blocker because a paradoxical increase in the ventricular rate may occur due to drug-induced conversion of AF to AFl. Propafenone is less prone to this phenomenon due to intrinsic β-adrenergic antagonism.

Sotalol is a class III antiarrhythmic agent that is useful for the maintenance of sinus rhythm. Sotalol is a mixture of stereoisomers (DL-); D-sotalol is a potassium channel blocker, while L-sotalol is a β-antagonist. Side effects reflect both mechanisms of action. In addition to QT interval prolongation leading to TdP, DL-sotalol may result in sinus bradycardia or AV conduction abnormalities. Sotalol should not be used in patients with decompensated CHF due to the negative inotropic effect or with a prolonged QT interval.

Dofetilide is a class III antiarrhythmic agent that is useful for the maintenance of sinus rhythm. Dofetilide blocks the rapid component of the delayed rectifier potassium current, IKr. As a result, dofetilide increases the QT interval at clinically effective doses. QT prolongation is intensified by bradycardia, a characteristic known as “reverse use dependence.” The main risk of dofetilide is TdP.

Dofetilide is contraindicated in patients with a baseline correct QT interval (QTc) >440 milliseconds, or 500 milliseconds in patients with bundle branch block. Initial dosing of dofetilide is based on the creatinine clearance.

A 12-lead ECG should be obtained before the first dose of dofetilide and 1 to 2 hours after each dose. If the QTc interval after the first dose prolongs by 15% of the baseline or exceeds 500 milliseconds, a 50% dosage reduction is indicated. If the QTc exceeds 500 milliseconds after the second dose, dofetilide must be discontinued.

Several medications block the renal secretion of dofetilide (verapamil, cimetidine, prochlorperazine, trimethoprim, megestrol, ketoconazole) and are contraindicated with dofetilide. The advantages of dofetilide are that it is not associated with increased CHF or mortality in patients with LV dysfunction , and dofetilide does not cause sinus node dysfunction or conduction abnormalities.

Amiodarone has the properties of class I, II, III, and IV drugs, and is arguably the most effective antiarrhythmic agent for maintenance of sinus rhythm. Because of the extensive toxicity profile of amiodarone, it should not be considered as a first-line agent for rhythm control of AF in patients in whom an alternative antiarrhythmic can be safely used.

Intravenous amiodarone has a low efficacy for acute conversion of AF, although conversion after several days of IV amiodarone has been observed. Given its common use and relative high incidence of side effects, a more detailed discussion of these effects is required.

DC cardioversion is the safest and most effective method of acutely restoring sinus rhythm. Prior to cardioversion, consideration of thromboembolic risk and anticoagulation is critical, when possible, to minimize thromboembolic events triggered by the cardioversion process.

AF with a rapid ventricular response in the setting of ongoing myocardial ischemia, MI, hypotension, or respiratory distress should receive prompt cardioversion regardless of the anticoagulation status.

If the duration of AF is documented to be <48 hours, cardioversion may proceed without anticoagulation. If AF has persisted for >48 hours (or for an unknown duration), patients should be anticoagulated with warfarin, with an international normalized ratio (INR) of 2.0 to 3.0, for at

least 3 weeks before cardioversion, and anticoagulation should be continued in the same therapeutic range following successful cardioversion.

An alternative to anticoagulation for 3 weeks before cardioversion is to perform a transesophageal echocardiogram to rule out left atrial appendage thrombus before cardioversion.

This method is safe and has the advantage of shorter time to cardioversion than warfarin and therefore is indicated in patients who are not able to wait weeks before cardioversion.

Therapeutic anticoagulation with warfarin is indicated after the cardioversion for a minimum of 4 weeks suggests that in patients with high risk for stroke, warfarin should be continued indefinitely.

When practical, sedation should be accomplished with midazolam (1 to 2 mg IV q2min to a maximum of 5 mg), methohexital (25 to 75 mg IV), etomidate (0.2 to 0.6 mg/kg IV), or propofol (initial dose, 5 mg/kg/hr IV).

Proper synchronization to the QRS is critical to avoid induction of ventricular fibrillation (VT) by a cardioversion shock delivered during a vulnerable period. Synchronization of the external cardioverter-defibrillator should be confirmed by noting the presence of a synchronization marker superimposed on the QRS complex.

For cardioversion of atrial arrhythmias, the anterior patch electrode should be positioned just right of the sternum at the level of the third or fourth intercostal space, with the second electrode positioned just below the left scapula posteriorly . Care should be taken to position patch electrodes at least 6 cm from PPM or defibrillator generators. If electrode paddles are used, firm pressure and conductive gel should be applied to minimize contact impedance. Direct contact with the patient or the bed should be avoided. Atropine (1 mg IV) should be readily available to treat prolonged pauses.

Junctional rhythms:

Junctional arrhythmias are often transient , occur during the initial two days characterized by narrow QRS complexes with retro grade P waves or AV dissociation.

There are two types of Junctional rhythms :

1. AV junctional rhythm at a rate of 35 to 60 beats/min in which the Av junctional tissue act as a pacemaker .

This arrhythmia is generally a benign protective escape rhythm commonly seen in the presence of inferior MI.

Transvenous sequential AV pacing may be required when there is hemodynamic impairment, to facilitatie ventricular performance and maintain adequate peripheral perfusion.

2. Acelerated junctional rhythm (nonparoxysmal junctional tachycardia) occurs when there is increased automaticity of the junctional tissue, , usually appearing at a rate of 70 to

130 beats/min, seen more commonly with inferior Acute MI and in digitalis intoxication.

Multifocal atrial tachycardia.

- Often a secondary rhythm associated with AMI ,
- Occurs in 5% to 7% of patients with AMI
- Multiform (at least three different P - wave morphologies)
- Rates of 100 per minute or more.
- The ventricular rhythm is irregular and rapid atrial
- Atrial activity, is well organized,
- Baseline is isoelectric between P waves.

MATERIALS AND METHODOLOGY

The study is conducted in government rajaji hospital madurai from july 2014 to October 2014 . A total of 100 patients admitted to the ICCU are selected to enter the study. This is a descriptive study.

Inclusion Criteria:

- 1. Patient in hyper-acute to acute phase of myocardial infarction and developed arrhythmia within first week of AMI based on third universal defenition of myocardial infarction.
- 2. Age above 19 years.

Exclusion Criteria:

1.The patients admitted with previous history of myocardial infarction

2. A known case of valvular heart diseases.

3. A known case of thyroid disorders.

4 .Patients with unknown and undefined arrhythmias of known premorbid condition.

Study Population:

A total of 100 patients are recruited on admission to the intensive coronary care unit at Government Rajaji Hospital. They included 74 males and 26 females. Patients with confirmed diagnosis of acute myocardial infarction and satisfying the inclusion and exclusion criteria are included in the study group.

The diagnosis of acute myocardial infarction is based on the Revised Definition of Myocardial Infarction.

Typical rise and gradual fall (troponin) or more rapid rise and fall (CK-MB) of biochemical markers of myocardial necrosis with at least one of the following:

a) Ischaemic symptoms.

b) Development of pathologic Q waves on the ECG reading.

c) ECG changes indicative of ischaemia (ST – segment elevation or depression).

d) Coronary artery intervention (eg: coronary angioplasty).

Clinical Data

A detailed history with special reference to the cardiovascular system is taken. A thorough physical examination is done with emphasis on the cardiovascular system.

Investigations

12-lead ECG was taken at admission, at 24 hours, 48 hours and at the time ofarrhythmia. Multi parameter monitors (AGE Medical Systems Company) was used to monitor the patients for 48 hours and the pattern of arrhythmias, if any, was noted . 2-D echocardiographic analysis was done wherever possible, during the first 48 hours of hospitalization.

Statistical Analysis

The current study is hospital – based descriptive study. The test of significance used between the associations of different characteristics was the Chi square test. For statistical significance, the p value was calculated and a value less than 0.05 was considered significant. SPSS11.5 was used to analyse the data.

+ Suggestive significance (P value: $0.05<P<0.10$)

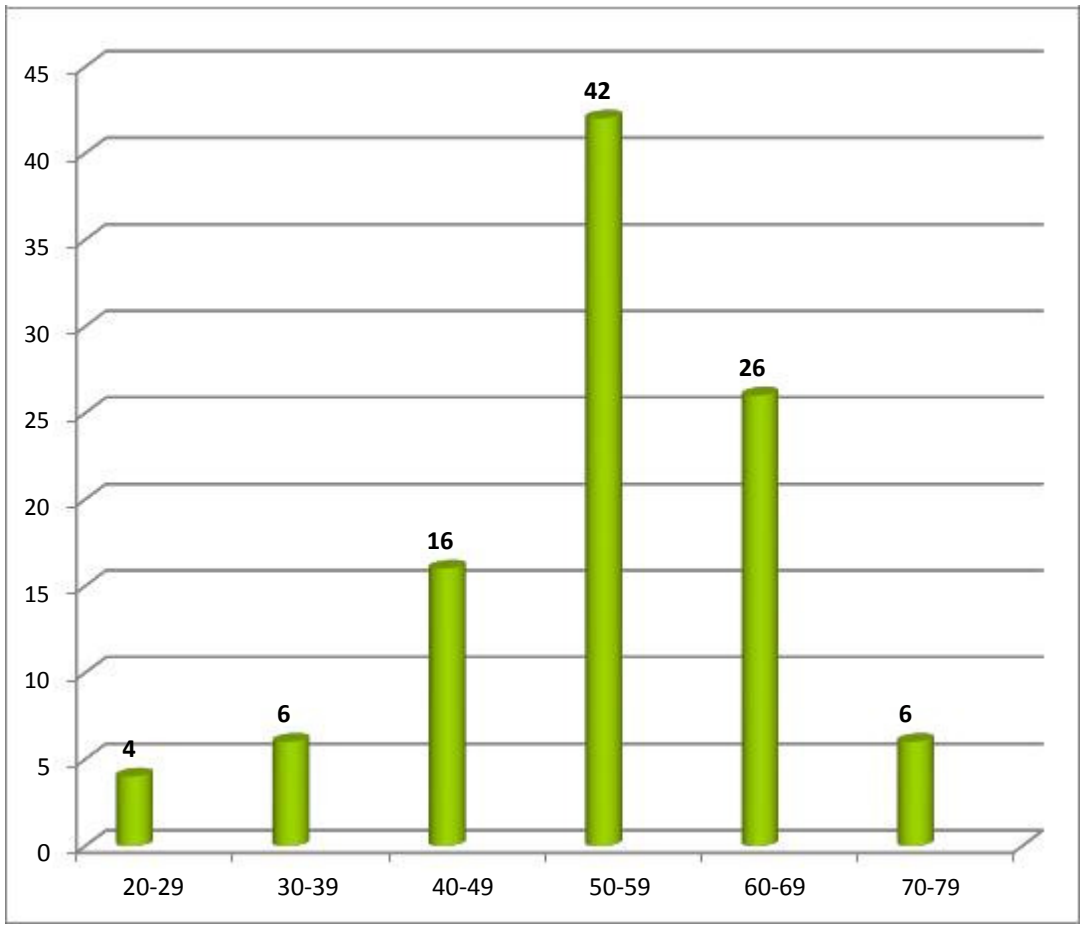
* Moderately significant (P value:$0.01<P \leq 0.05$)

** Strongly significant (P value : $P \leq 0.01$).

OBSERVATIONS AND RESULTS

A total number of 100 patients were studied

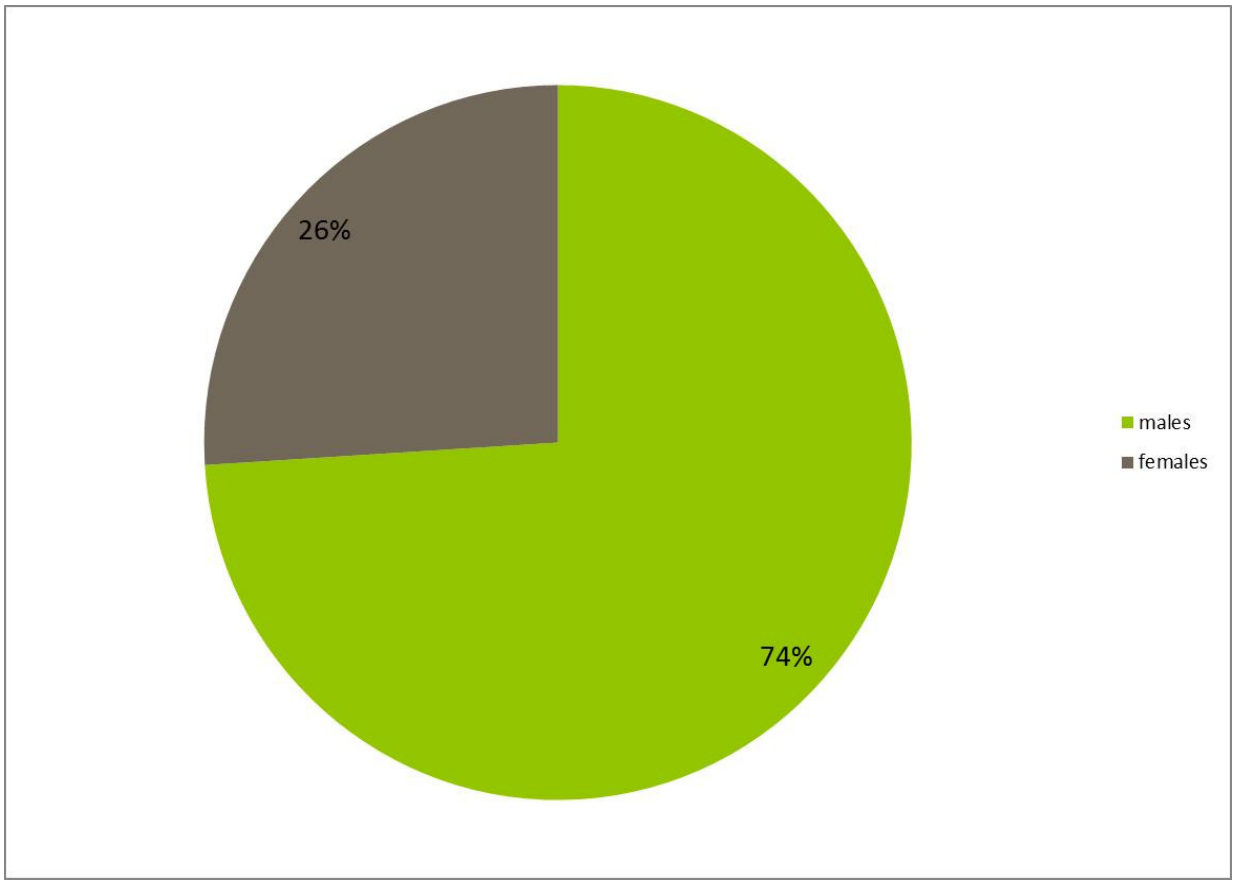
Age distribution of cases



The study group with 100 patients had ages ranging from 20 to 80 years .

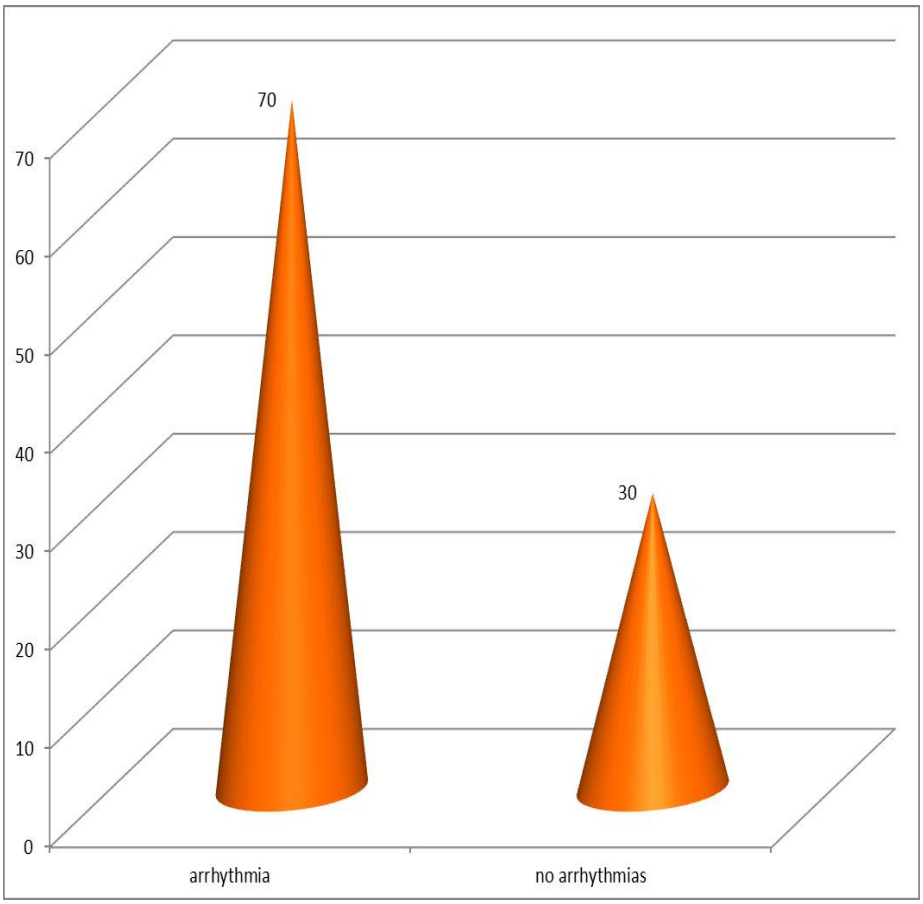
majority of patients were above the age of 50 years .

Gender distribution of cases



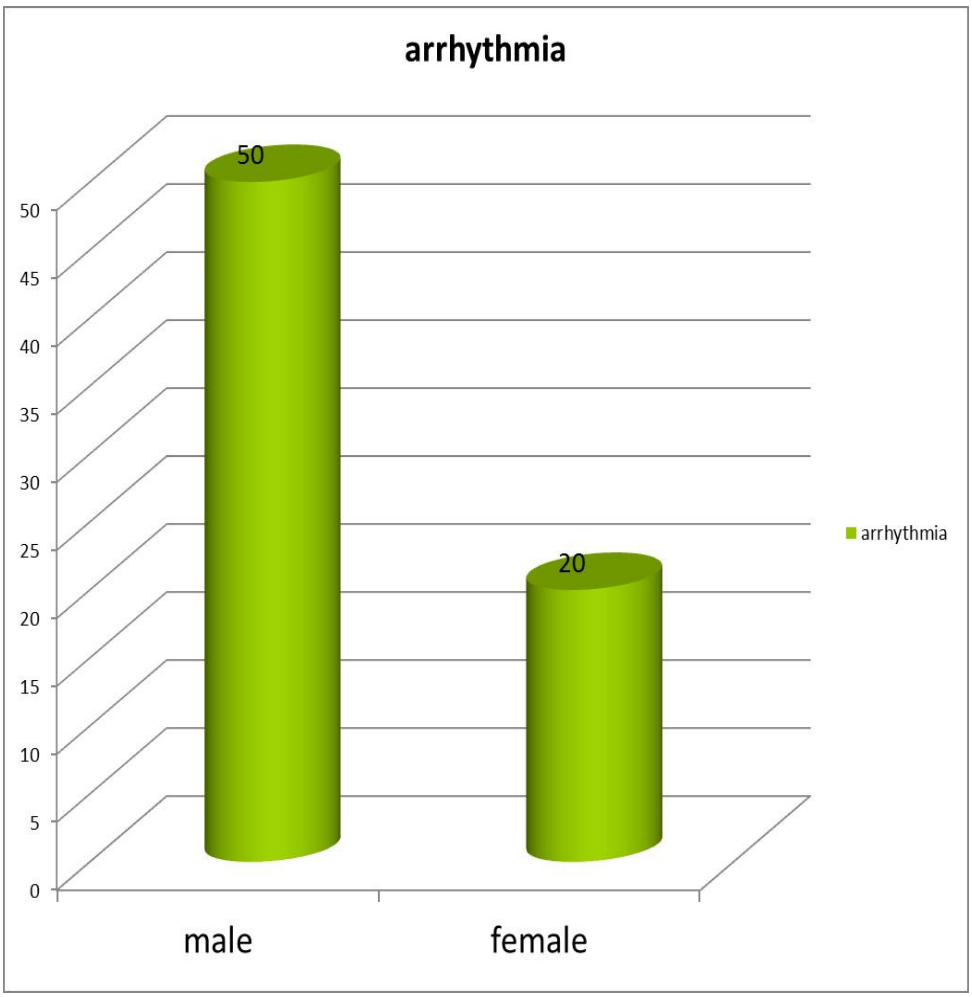
74% of patients were males and 26% by females.

Occurance of Arrhythmia



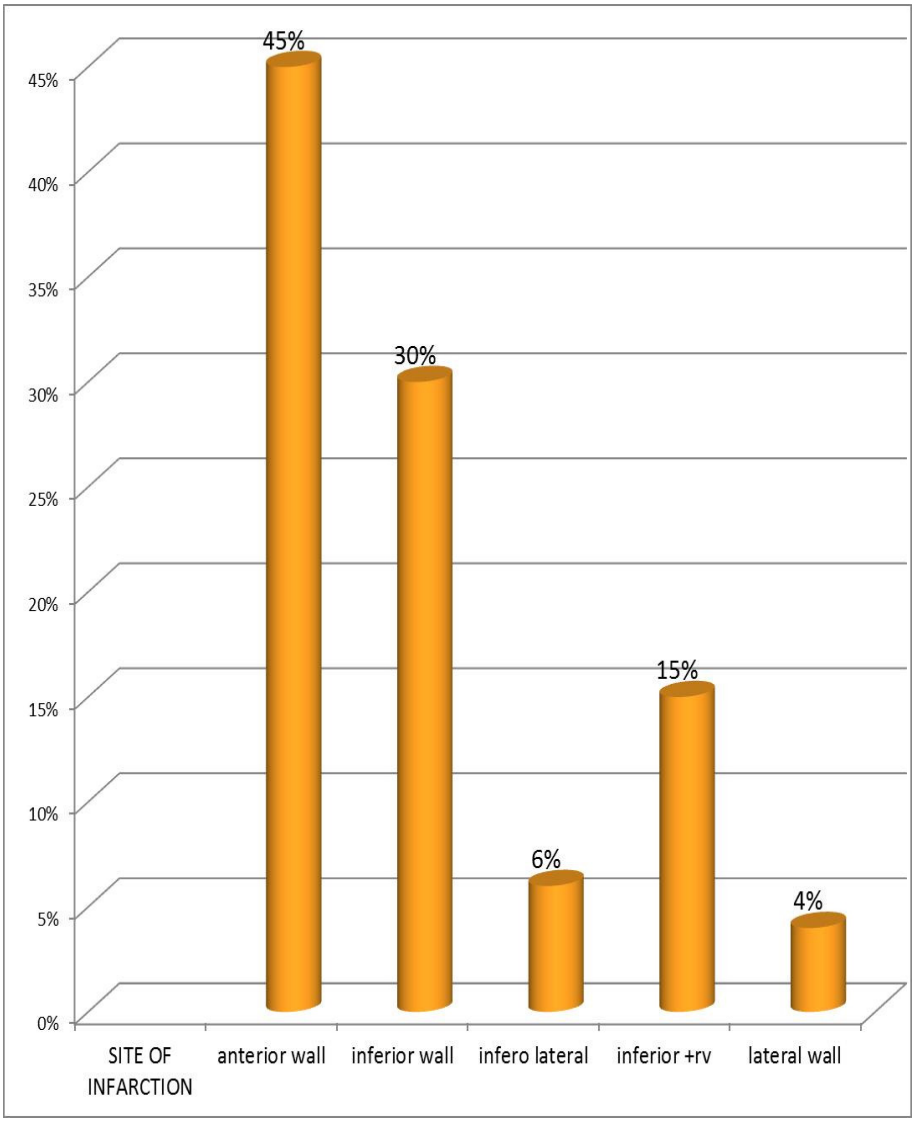
Out of 100 patients , 70 patients had arrhythmias.

Arrhythmias sex distribution



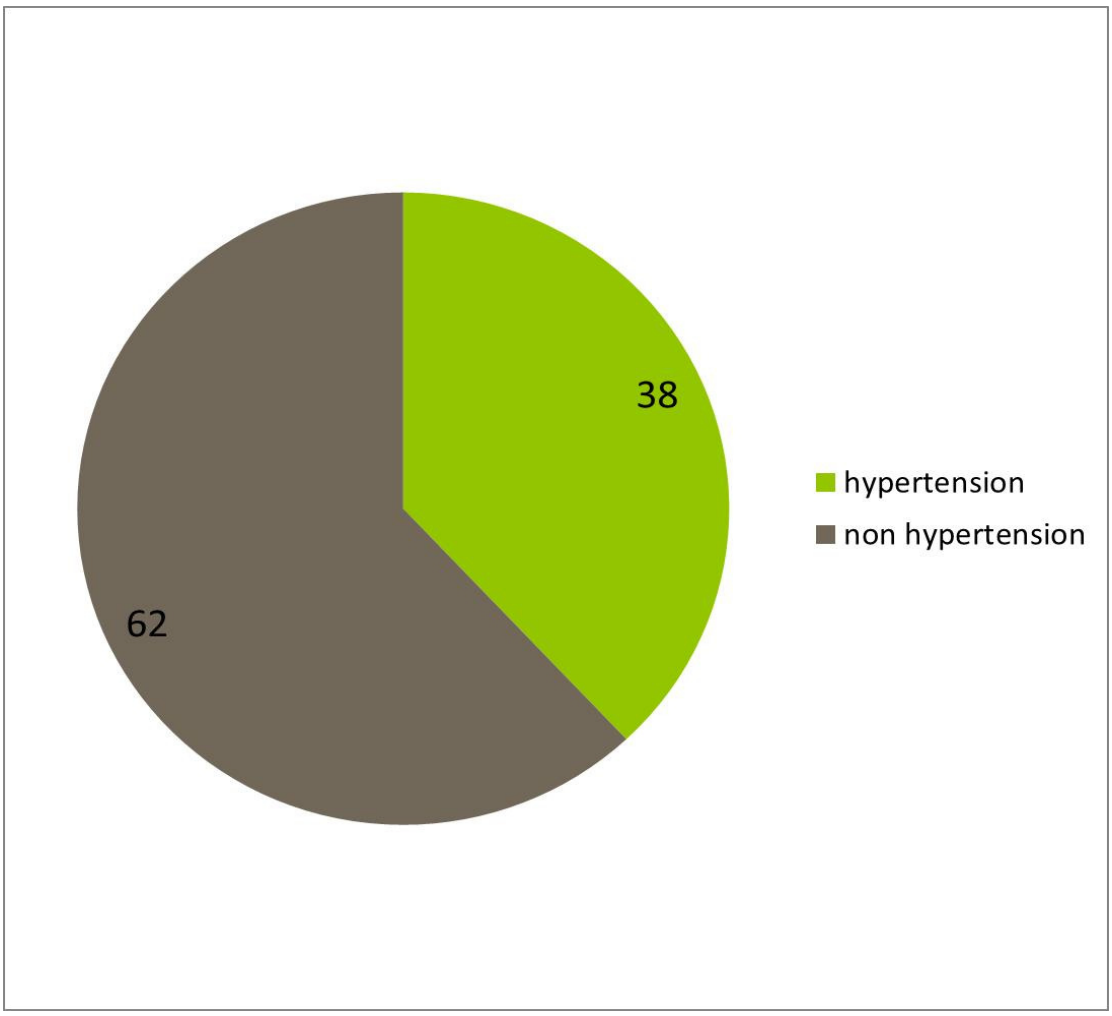
Arrhythmia occurred in 77% of females and in 67.5% of males.

SITE OF INFARCTION



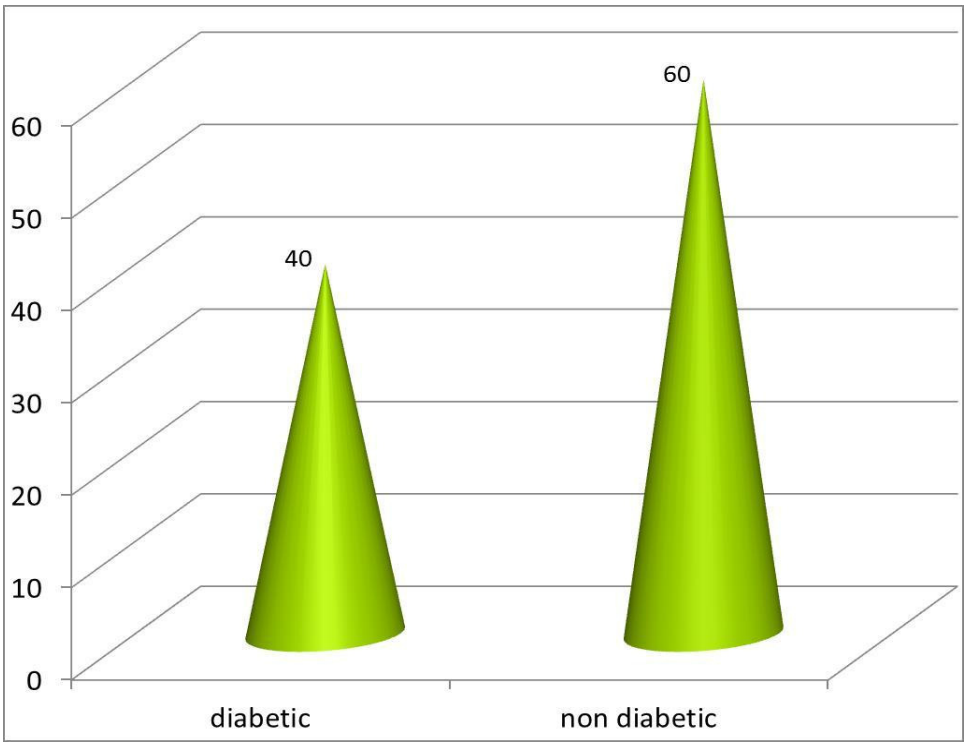
Majority of patients had anterior wall infarction , followed by inferior wall infarction.

Hypertensive vs non hypertensive:



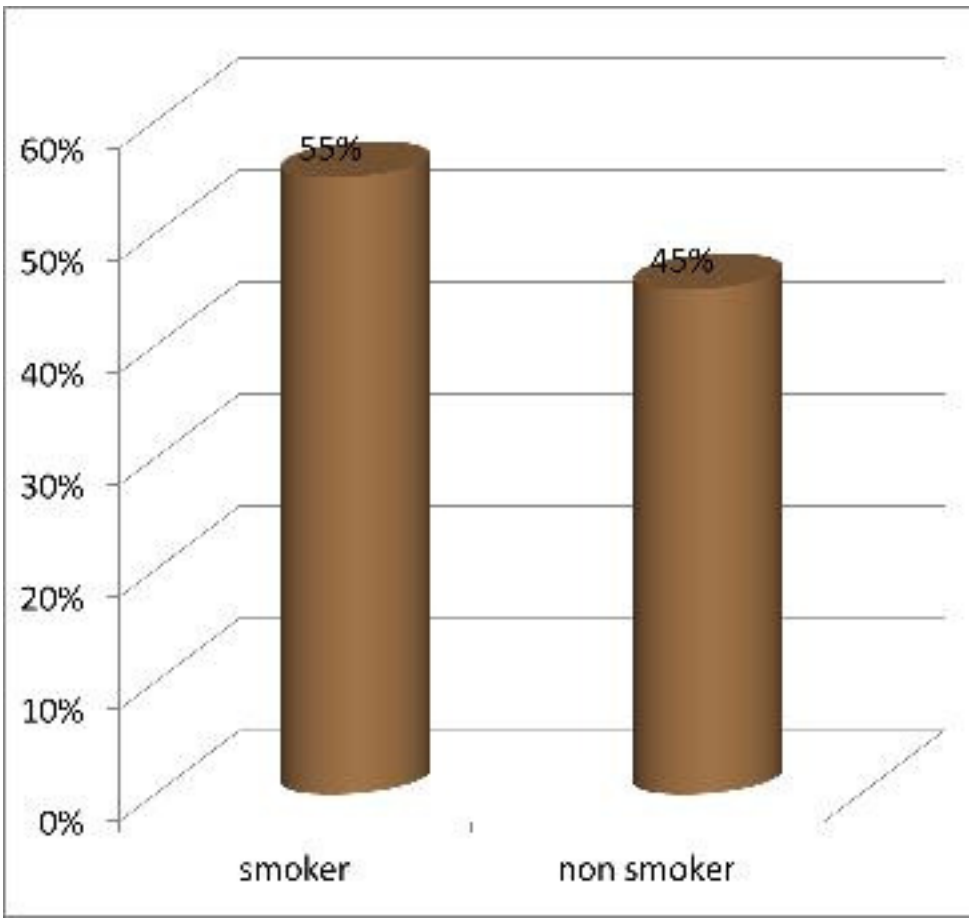
Majority of patients were non hypertensives.

Diabetic vs non diabetic:



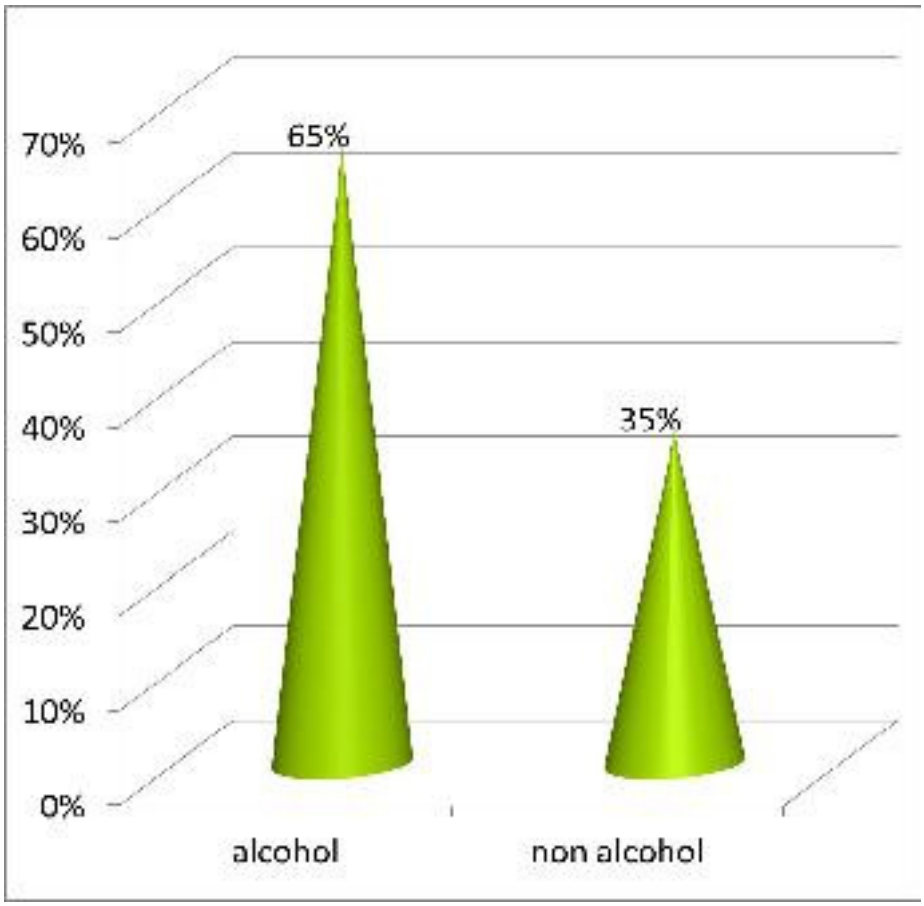
Out of 100 patients 40 patients were diabetics.

Smoker vs non smoker



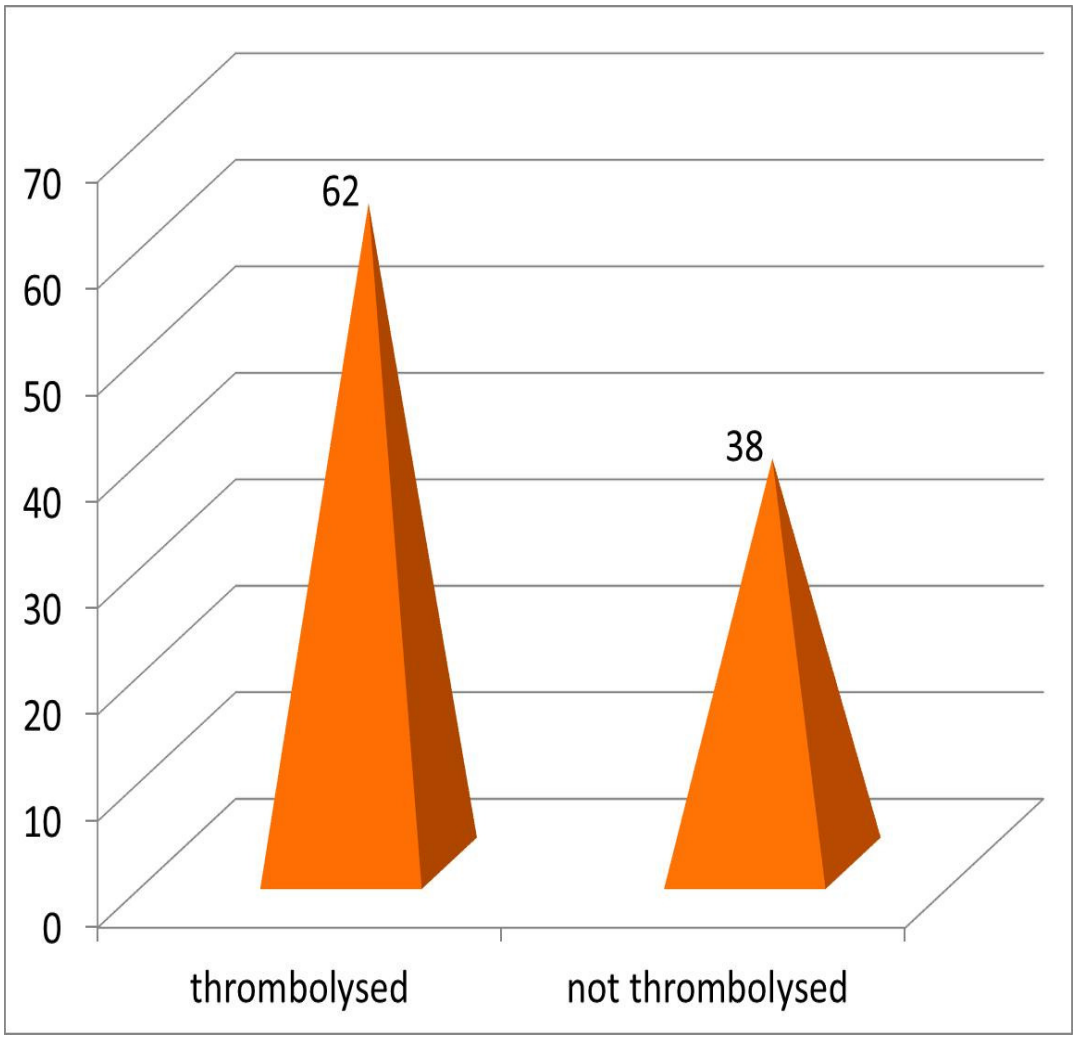
Majority of patients were tobacco smokers.

Alcohol vs non alcohol:



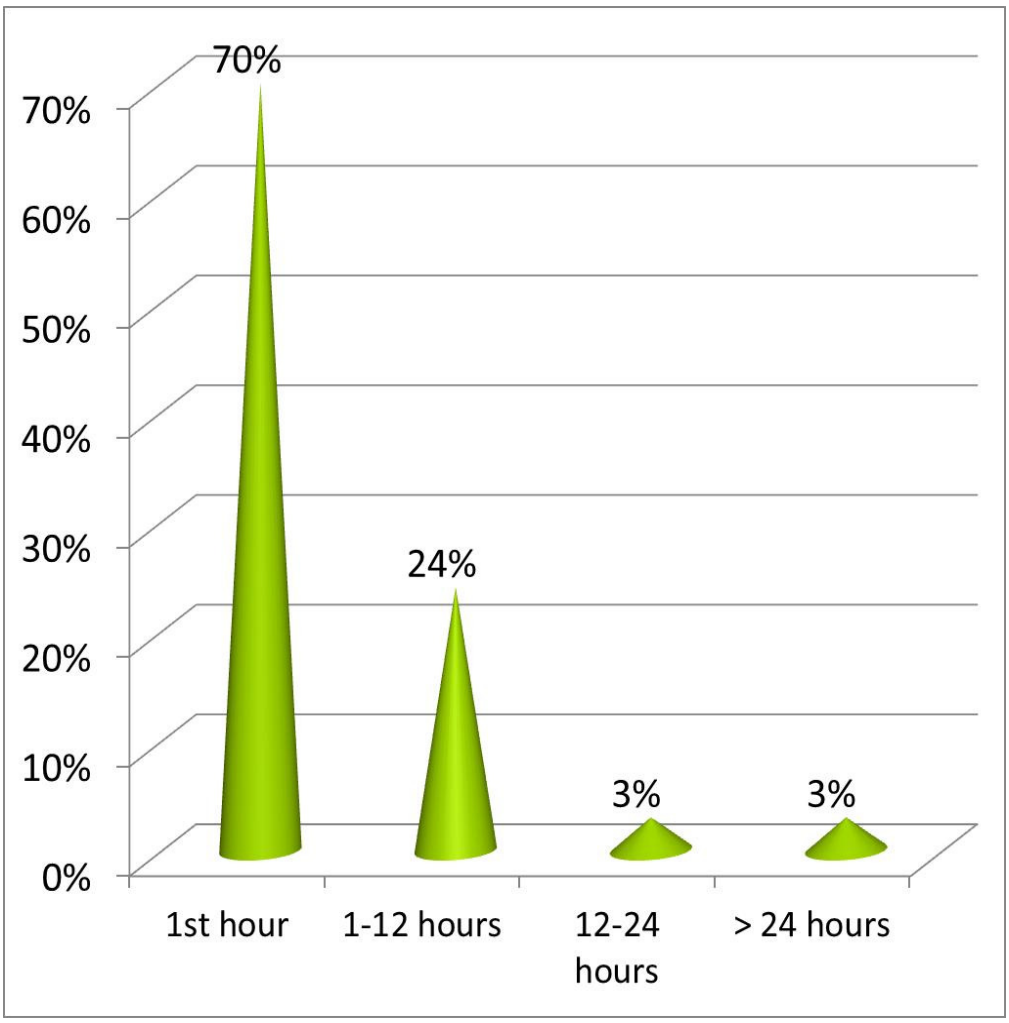
About 2/3rd of patients consumed alcohol.

THROMBOLYSIS TREATMENT



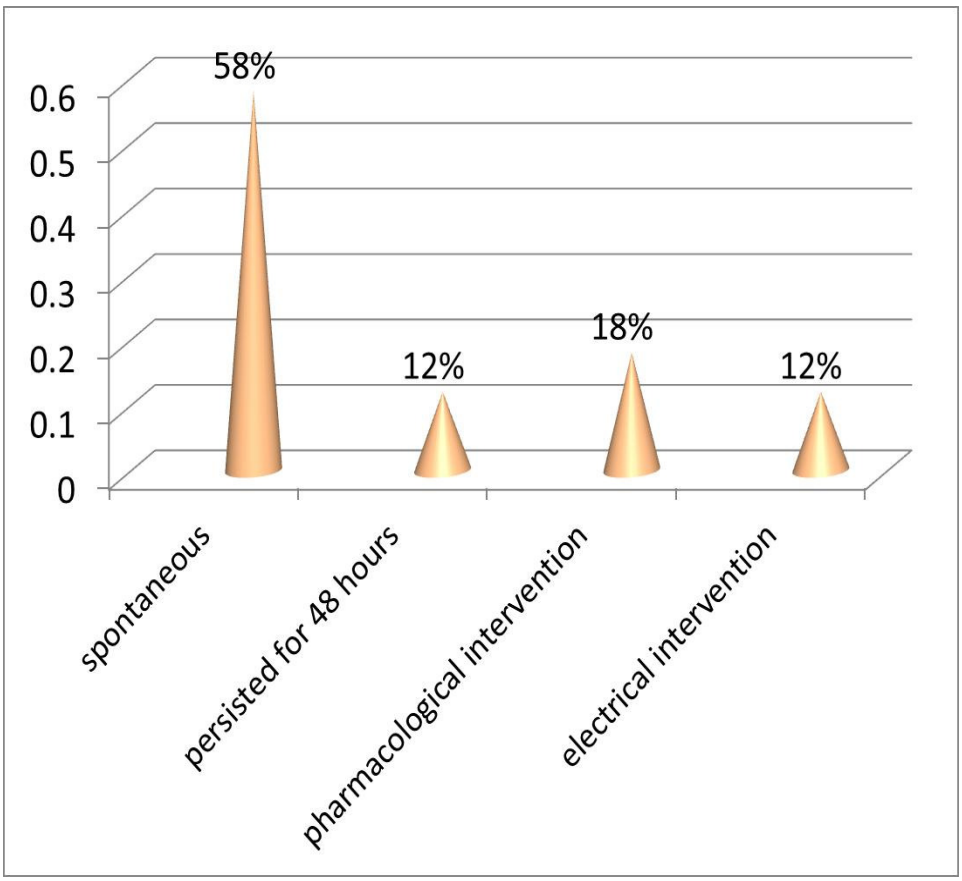
Out of 100 patients 62% patients were thrombolysed.

Time of arrhythmia



Majority of arrhythmias occurred during the first hour of hospitalization and 1/4th of arrhythmia occurred during 1-12 hours.

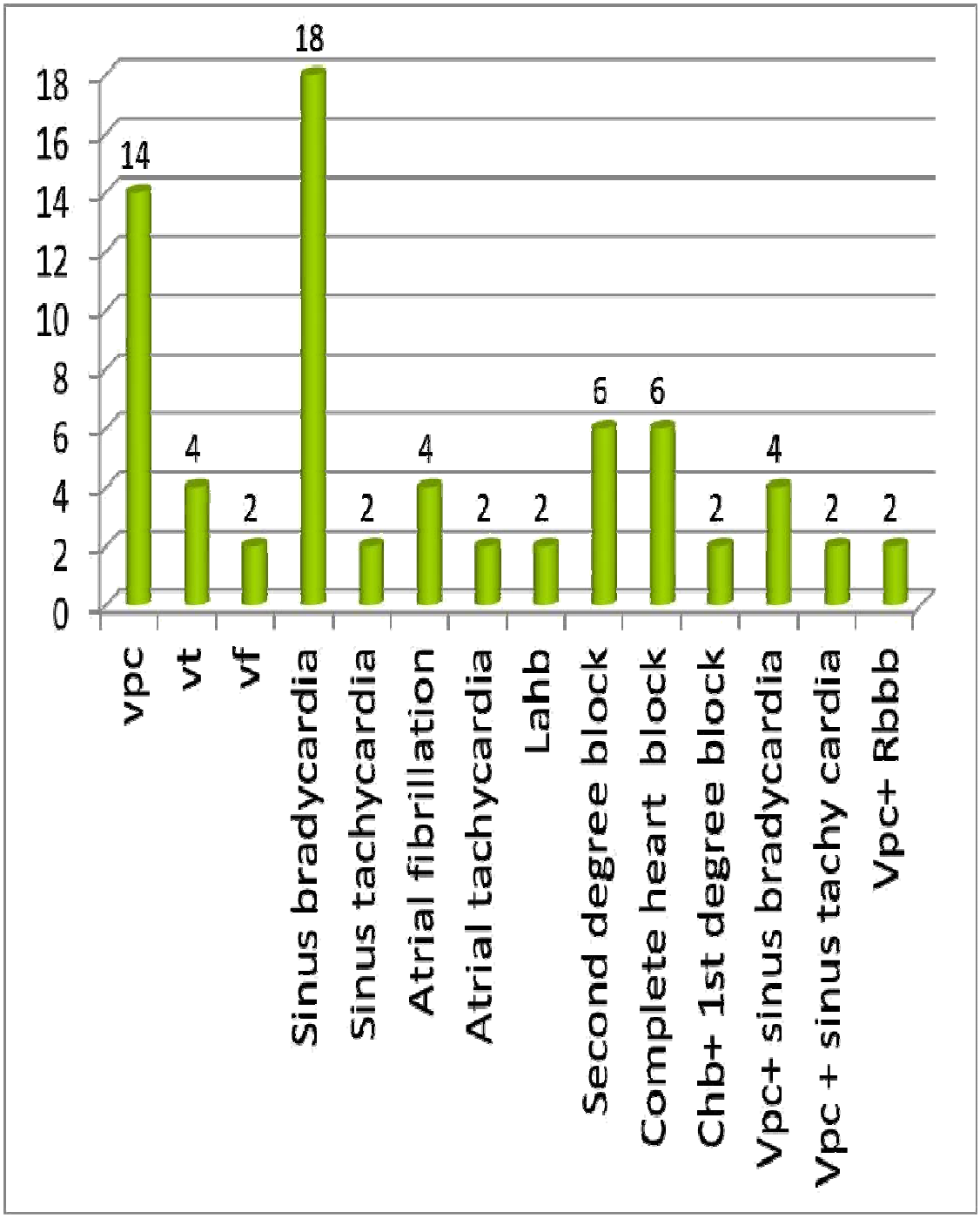
Mode of termination of Arrhythmia



Majority of arrhythmias underwent spontaneous resolution. It persisted in 12% of patients for 48 hours ,18% required pharmacological intervention ,12% required electrical intervention.

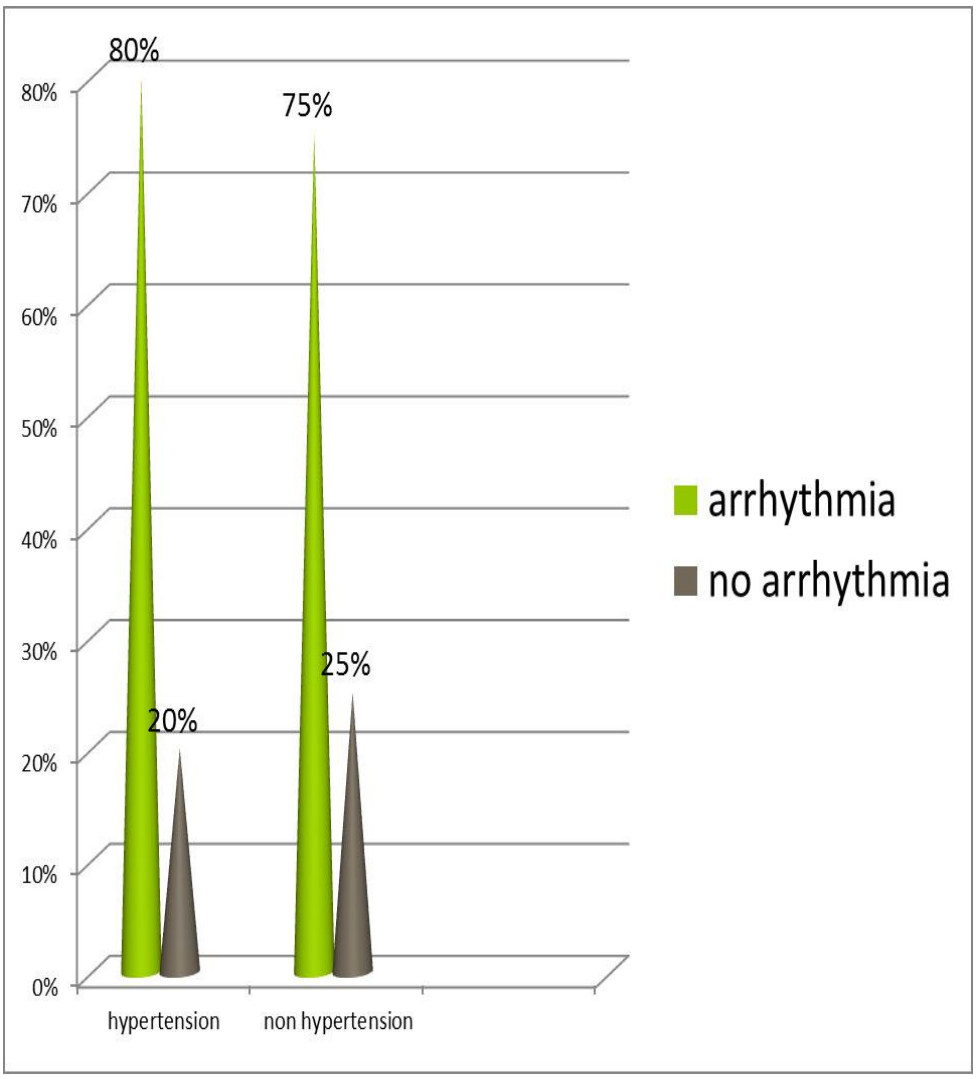
Specific arrhythmias

TYPE OF ARRHYTHMIA	FREQUENCY	PERCENTAGE
Vpc	**14**	**20%**
Vt	**4**	**5.71%**
Vf	**2**	**2.85%**
Sinus bradycardia	**18**	**25.71%**
Sinus tachycardia	**2**	**2.85%**
Atrial fibrillation	**4**	**5.71%**
Atrial tachycardia	**2**	**2.85%**
Lahb	**2**	**2.85%**
Second degree block	**6**	**8.57%**
Complete heart block	**6**	**8.57%**
Chb+ 1st degree block	**2**	**2.85%**
Vpc+ sinus bradycardia	**4**	**5.71%**
Vpc + sinus tachycardia	**2**	**2.85%**
Vpc+ Rbbb	**2**	**2.85%**



Sinus bradycardia was the most common arrhythmia. VPCs occurred in isolation in 14% of patients and it also occurred along with other arrhythmia.

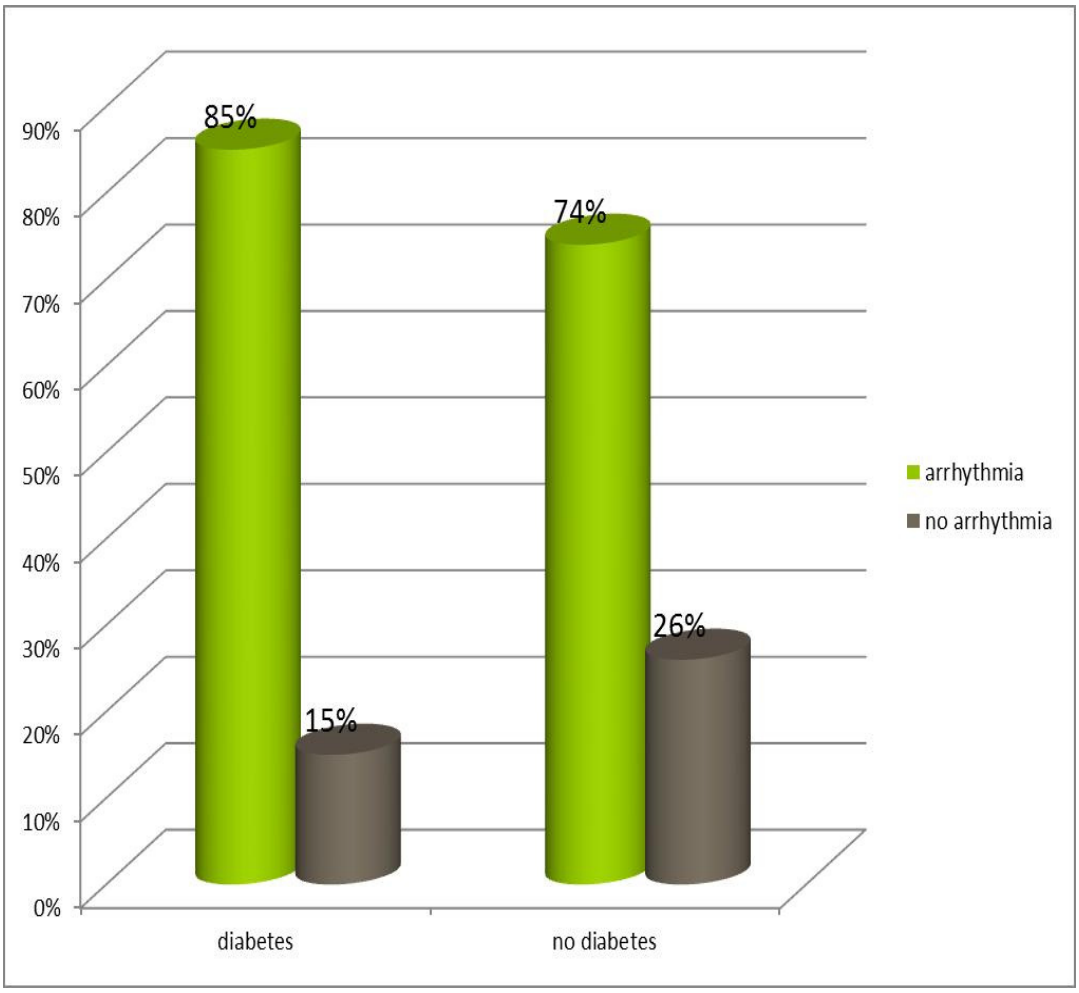
Hypertension vs arrhythmia



P=0.O13

80% of hypertensives were detected to have arrhythmia. It was statistically significant.

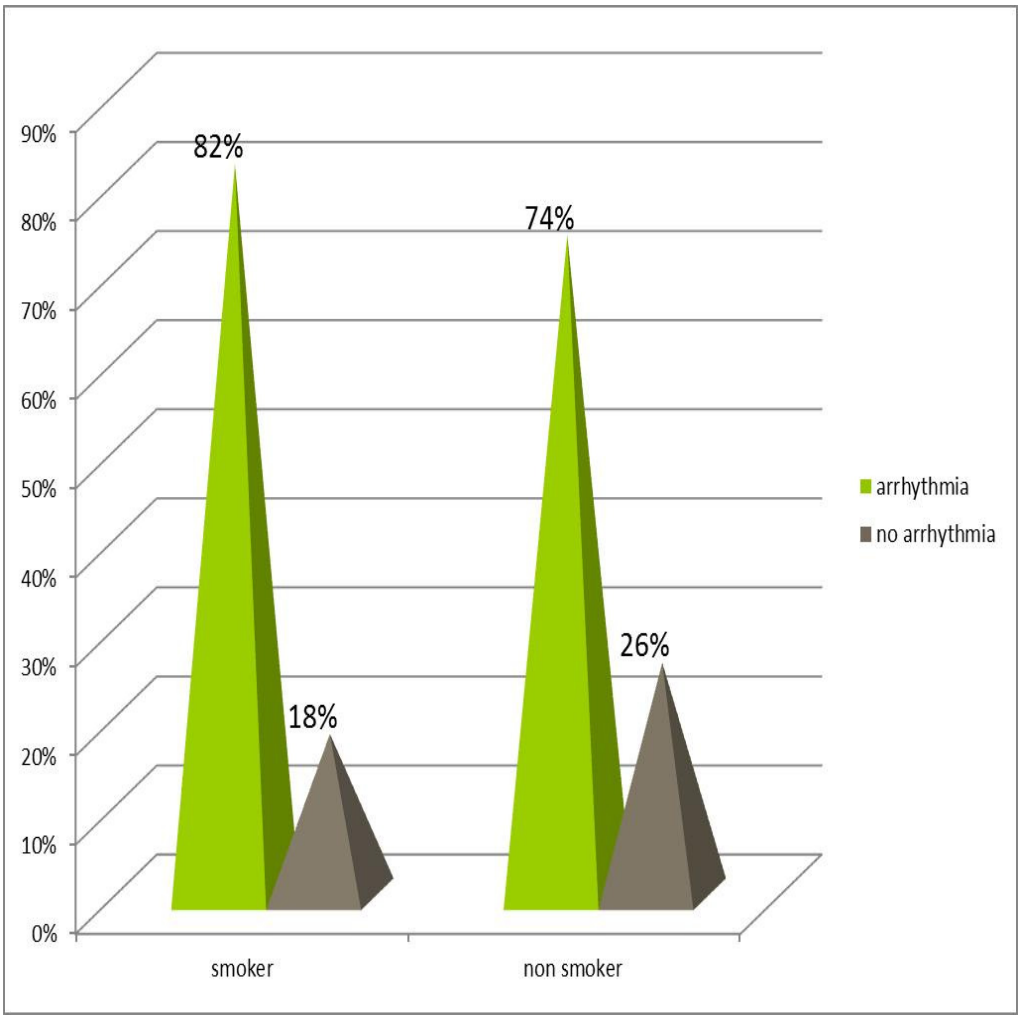
Diabetes vs arrhythmias



P=O.OO3

85% of those with diabetes mellitus had arrhythmia and found to have statistically significant.

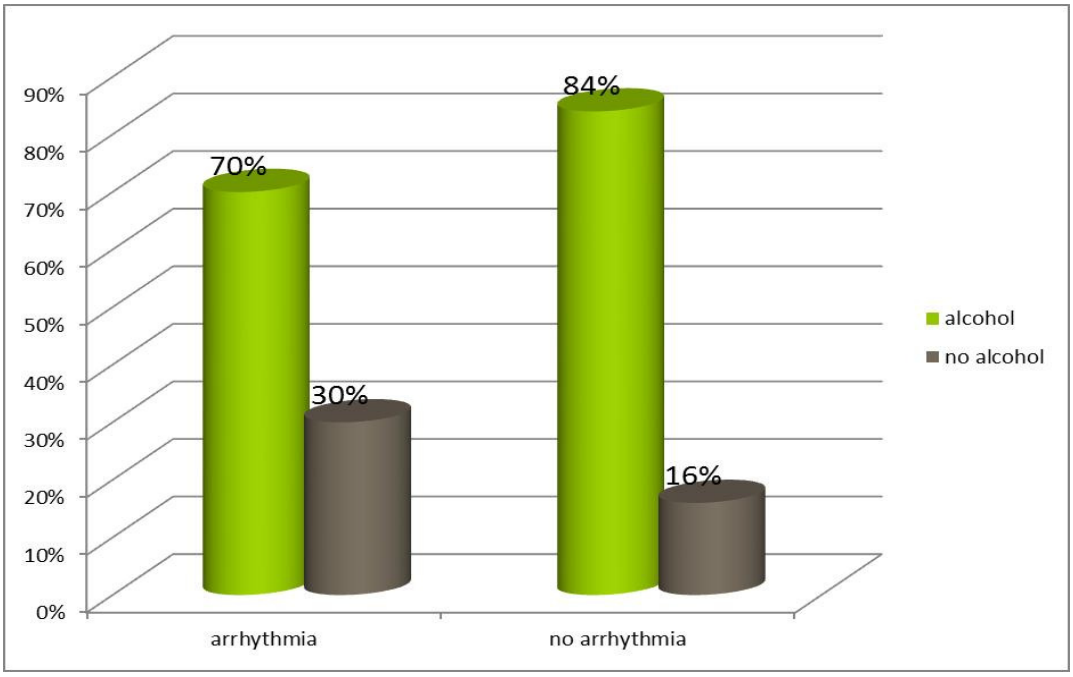
Smoking vs arrhythmia



P=0.004

82% of smokers had arrhythmia, it was statistically significant.

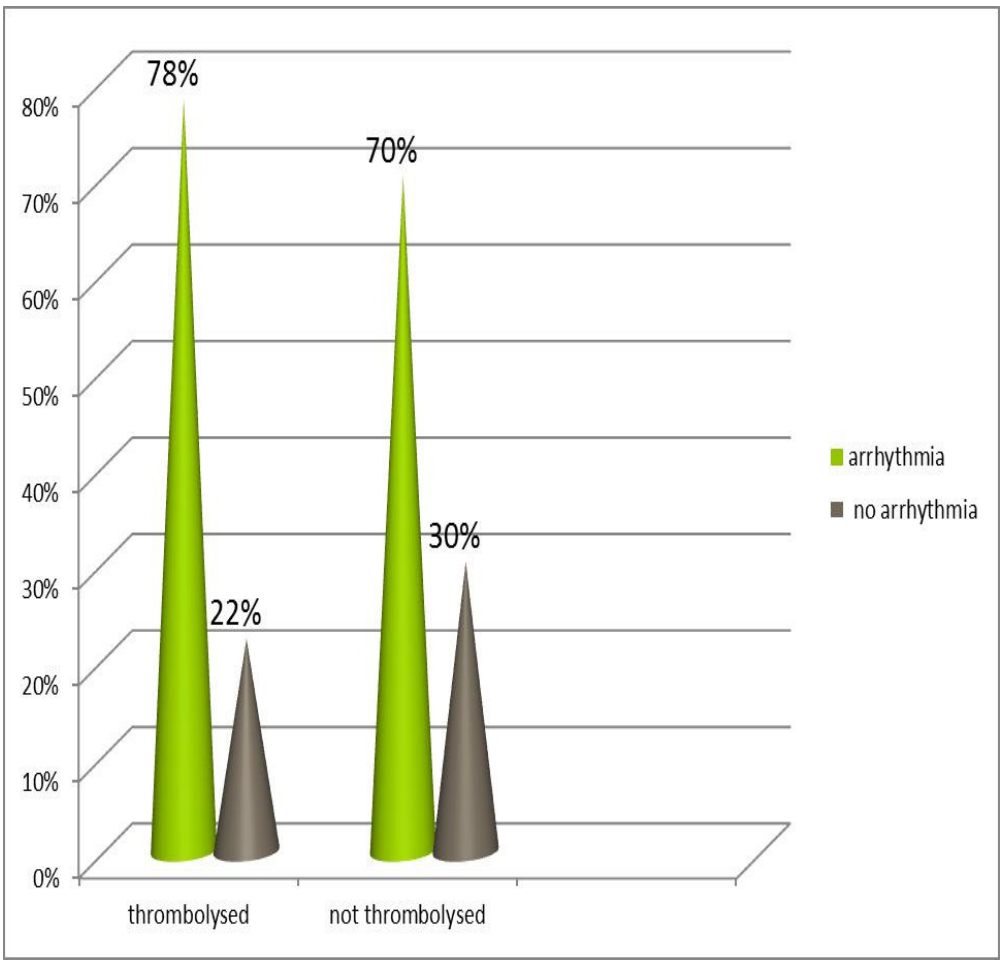
Alcohol vs arrhythmia



P=0.OO2

70% of those consuming alcohol had arrhythmia and was statistical significant.

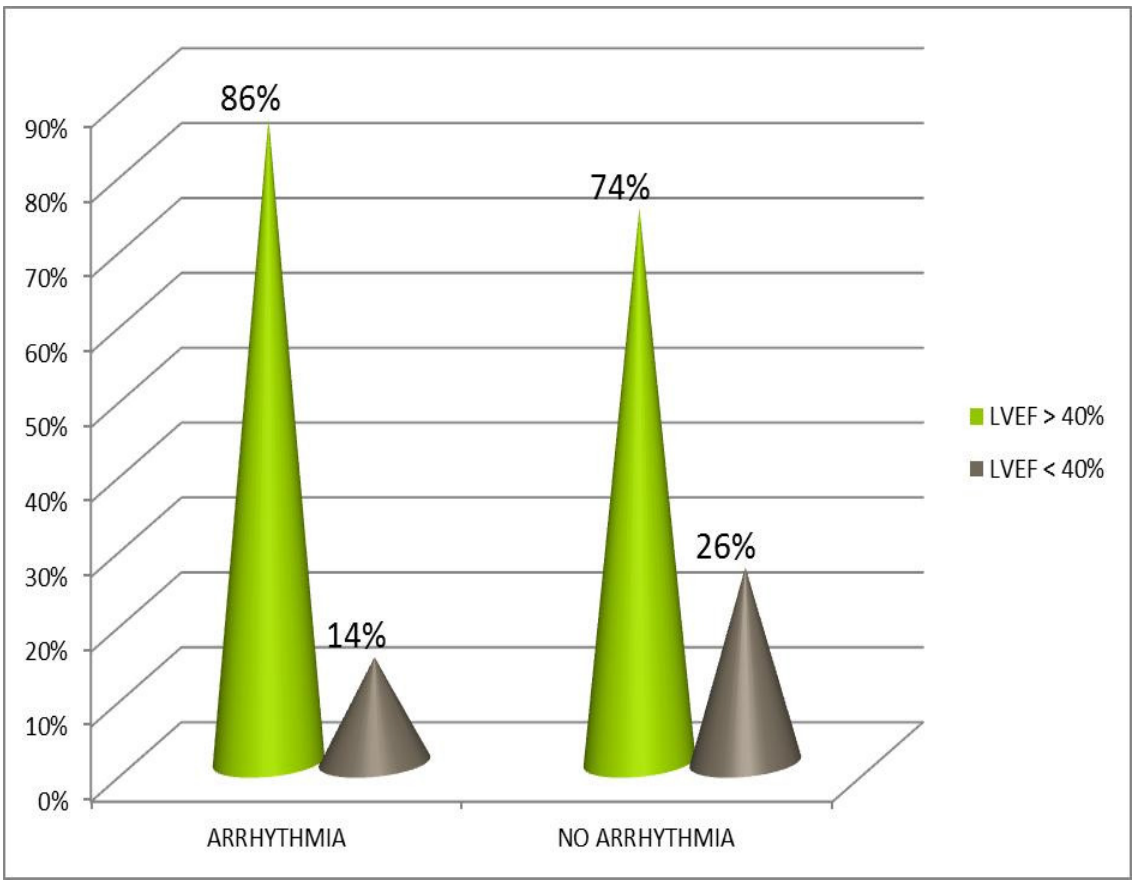
Arrhythmia vs thrombolysis



P=0.035.

Arrhythmia occurred more in those who were thrombolysed than in those who were not thrombolysed .It was statistically significant.

Left ventricular ejection fraction vs arrhythmia



86% of of patients with LVEF > 40 % had arrhythmia . It was not statistically significant.

SITE OF INFARCTION VS LV DYSFUNCTION

Type of infarction	Lvef < 40 %	Lvef > 40%
Anterior wall mi	70%	30%
Inferior wall mi	23%	77%
Infero lateral mi	34%	66%
Inferior wall+ right ventricular mi	100%	-
Lateral wall mi	58%	42%

In AWMI majority of patients in whom 2-D ECHO was done,had L.V. dysfunction. In IWMI majority did not have L.V. dysfunction.

Results and interpretation :

A total of 100 patients are recruited on admission to the intensive coronary care unit at Government Rajaji Hospital. They included 74 males and 26 females. Patients with confirmed diagnosis of acute myocardial infarction and satisfying the inclusion and exclusion criteria are included in the study group.

This study showed myocardial infarction was more common among elderly .

In this study myocardial infarction was common among males than females.

In this present study Out of 100 patients , 70 patients had arrhythmias.

In this present study Arrhythmia occurred in 77% of females and in 67.5% of males.

In this present study anterior wall MI occured in 45% of patients ,30% of patients had inferior wall MI ,15% patients had inferior wall and right ventricular MI ,6% of patients had infero lateral MI ,4% of patients had lateral wall MI .

In this present study 38% of patients were hypertensive and 62% of patients were non hypertensives.

In this present study 40% of patients were diabetics and 60% of patients were non diabetics.

In this present study 55%% of patients were smokers and 45% of patients were non smokers .

In this present study 65%% of patients were alcoholics and 35% of patients were non alcoholics .

In this present study 62 % of patients were thrombolysed and 38% of patients were not thrombolysed .

In this present study 70 % of arrhythmia occured during the 1st hour ,24% of patients had arrhythmias during 1 to 12 hours ,3 % of patients had arrhythmias during 12 to 24 hours .

82% of smokers had arrhythmia, it was statistically significant.

70% of those consuming alcohol had arrhythmia and was statistical significant.

Arrhythmia occurred more in those who were thrombolysed than in those who were not thrombolysed .It was statistically significant.

86% of of patients with LVEF > 40 % had arrhythmia . It was not statistically significant.

In AWMI majority of patients in whom 2-D ECHO was done,had L.V. dysfunction. In IWMI majority did not have L.V. dysfunction.

Frequency of arrhythmias :

Vpc - 20% , Vt - 5.71%, Vf - 2.85%

Sinus bradycardia - 25.71%

Sinus tachycardia - 2.85%

Atrial fibrillation - 5.71%

Atrial tachycardia - 2.85%

Lahb - 2.85%

Second degree block - 8.57%

Complete heart block - 8.57%

Chb+ 1st degree block - 2.85%

Vpc+ sinus bradycardia - 5.71%

Vpc + sinus tachycardia - 2.85%

Vpc+ Rbbb - 2.85%

DISCUSSION

The current study is a descriptive study and included 100 patients.

Patients were evaluated with special reference to the pattern of cardiac arrhythmias in acute myocardial infarction during the first week of hospitalization.

Studying arrhythmias in hospitalized cases of acute myocardial infarction is an indirect estimate of mortality and assumes significance because true mortality due to acute myocardial infarction is difficult to ascertain in the community due to inadequate reporting and low autopsy rates.

Indian show higher incidence of mortality than other ethnic groups.

Also, South Indians have higher prevalence.

The conventional risk facter namely age, sex, hypertension, diabetes mellitus, smoking and alcohol were also evaluated in these patients.

This study showed myocardial infarction was more common among elderly, in accordance to the American Heart Association observationAlso,

This study showed myocardial infarction was more common among elderly, in accordance to the American Heart Association observation.

In the study by SZ Abildstrom et al105 as compared to non-sudden cardiac death, the risk of sudden cardiac death, is relatively highest in the younger age groups, but the absolute risk of sudden cardiac death, is much higher among the upper age groups than the younger..

In the present study arrhythmia was detected in 70% of the patients. In a study by Aufderheide TP2 , 90% of patients with acute myocardial infarction have some cardiac rhythm abnormality during the first 24 hours following infarct onset.

The present study majority of arrhythmias occurred during the first hour of hospitalization. In the study by Aufderheide TP2 , approximately 25% have cardiac conduction disturbance within 24 hours following infarct onset.

This study showed a male preponderance as was observed in the Framingham Heart study.

In a prospective community based study by Shmuel Gottlieb et al1 0 7 of consecutive AMI patients hospitalised in CCUs in the mid 1990s indicate that women fare significantly worse than do men at 30 days. In a study by Yee Guan Yap et al1 0 8 in high – risk post – MI patients with LVEF <40% or frequent VPCs, the risk of arrhythmia deaths was higher than that of non arrhythmia deaths for up to two years although in female patients, they became increasingly more likely to die from non arrhythmic deaths after 6 months.

The risk of sudden cardiac death, following myocardial infarction was slightly lower in women.

The Framingham study106 demonstrated that smokers have a 2-3 fold increase in sudden cardiac death in each decade of life at entry between 30 and 50 years and that this is one of the few risk factors in which the proportion of CAD deaths that are sudden increases in association with the risk factors.

However in our study n statistically significant association was found between smoking and the occurrence of arrhythmia.

In a study by Hallstrong AP et al109, out of 310 survivors of out of the hospital cardiac arrest, the recurrent cardiac arrest rate was 27% at 3 year follow-up among those continued to smoke after the index event compared with 19% in those who stopped smoking.

In the present study VPCs were observed in 14% of the patients when they occurred alone. However they also occurred in the same patient along with other arrhythmias like heart blocks and tachyarrhythmias.

In a study by Campbell RW etal and Bigger JT et al , VPCs of various frequencies were observed in upto 90% of patients with MI.

In a study by Volpi A. et al , approximately 36% of patients with acute myocardial infarction presented with less than one premature

ventricular beat per hour in Holter, whereas almost 20% of patients showed frequent (more than 10 premature ventricular beats per hour).

In a study by Volpi A. et al41 , approximately 36% of patients with acute myocardial infarct ion presented with less than one premature ventricular beat per hour in Holter, whereas almost 20% of patients showed frequent (more than 10 premature ventricular beats per hour).

. In a study by Irwin JM58, sinus tachycardia was observed in upto 30% of the patients. In the present study, VT occurred alone in 5.1% of the patients.

It also occurred along with sinus tachycardia in 2.5% of patients and along with other ventricular arrhythmias in 2.5% of the patients. In a study by Echt DS et al and the CAST investigators, 20% of patients had nonsustainedVT and only 10% had more than one run of VT in 24 hours.

In a study by Tofler GH et al44 , sustained VT occurring within 48 hours of MI seen in 2% of patients is often transient and is not associated with long –term risk of sudden cardiac death.

In a study by Wolfe CL et al28, polymorphic VT seen in 2% of patients with MI is often rapid, symptomaticand hemodynamically and electrically unstable.

In our study, all the patients with VT had L.V. dysfunction. In a study by Bigger JT et al110 ,nonsustained VT is linked to an increased risk of sudden death during the first 6 to 12 months after MI especially hen associated with reduced LVEF (<40%). In our study VT was present along with VF in 2.1% of the patients.

In a study by Newby KH et al30, sustained VT and VF occur in upto 20% of patients with AMI and have been associated with poor prognosis.

In the present study, sinus tachycardia occurred in 2.85% of the patients .

In the present study, VT occurred alone in 5.71% of the patients. It also occurred along with sinus tachycardia in 2.5% of patients and along with other ventricular arrhythmias in 2.5% of the patients.

In a study by Echt DS et al and the CAST investigators, 20% of patients had nonsustainedVT and only 10% had more than one run of VT in 24 hours.

In our study, all the patients with VT had L.V. dysfunction. In a study by Bigger JT et al110 ,nonsustained VT is linked to an increased risk of sudden death during the first 6 to 12 months after MI especially when associated with reduced LVEF (<40%).

In the present study 8.57% of the patients had complete heart block and was associated with L.V dysfunction.

In the present study, ventricular fibrillation occurred in 2.85% of the patients.

In a study by Tofler GH et al , the incidence of ventricular fibrillation is highest during first 24 to 48 hours, particularly within the first 4 hours after the acute event, and may occur in upto 5% of patients.

In the present study, sinus bradycardia was observed in 25.71%% of the patients. In a study by Pantridge J.F et al , sinus bradycardia was observed in 25 to 40 percent of the patients.

In the present study, LAHB was seen in 2.85% of the patients, second degree heart block 8.57% of the patients, CHB along with first degree heart block was seen in 2.85% of the patients ,RBBB was present along with VPC in 2.85% of the patients.

Complete heart block is seen in 5% to 8% of patients with AMI and generally occurs early in the course of infarction.

A study by Goldberg RJ et al54 , showed that in hospital mortality is significantly higher with anterior wall infarction with complete heart block than with inferior wall myocardial infarction and that complete heart block is twice as common with inferior or posterior wall infarction as with anterior wall involvement.C omplete heart block is seen in 5% to 8% of patients with AMI and generally occurs early in the course of infarction.

Archbold RA et al63 observed complete heart block in 5.3% and complete heart block involving both bundle branches in 1.6%, bifascicular block in 2.9%. More advanced degree of block in patients with diabetes, AWMI, LVF, previous infarction, Q wave infarction.

In our study majority of patients with heart block had L.V. dysfunction. Alan S Go et al73 , noted conduction defects in 16%

of cases and concluded that a small decline in rate of severe conduction defects compared with previous studies, possibly reflecting the beneficial effects of thrombolytic therapy on infant size.

In the present study, 12% of patients had IWMI + RV. Simon H. Braet et al1 1 2 concluded that the incidence of high degree AV nodal block in patients with RV involvement was 48% compared to only 13% in patients with IWMI without RV involvement.

Results from TIMI II57 , showed that heart - block occurred in 12%, 63% had on presentation, 5.7% in 24 hours after treatment with rt –PA and patients with heart block at entry were old and had greater proportion of cardiogenic shock. Heart -block is common among patients with inferior infarction given thrombolytic therapy and is associated with increased mortality.

In a study by Harpaz D et al56 , the incidence of complete heart block complicating AMI is lower in the thrombolytic era than in the prethrombolytic era.

The AMI patients who develop complete heart block in the thrombolytic era have significantly worse prognosis than do patients without complete heart block.

In the present study, 15%% of patients had IWMI + RV. Simon H. Braet et al concluded that the incidence of high degree AV nodal block in patients with RV involvement was 48% compared to only 13% in patients with IWMI without RV involvement. Results from TIMI II , showed that heart - block occurred in 12%, 63% had on presentation, 5.7% in 24 hours after treatment with rt –PA and patients with heart block at entry were old and had greater proportionof cardiogenic shock.

Heart -block is common among patients with inferior infarction given thrombolytic therapy and is associated with increased mortality.

In the current study, atrial tachycardia occurred in 2.85% of patients and atrial fibrillation in 5.71%. In a study by Jewitt DE, et al113 , atrial arrhythmias occur in upto 20% within 24 hours of infarction.

In the present study 100% of those with atrial tachycardia had L.Vdysfunction. .

In a study by Jewitt DE, et al113 , atrial arrhythmias occur in upto 20% within 24 hours of infarction.

The SPRINT Study Group114 and Goldberg RJ et al115 , observed that atrial fibrillation is seen in upto 15% of patients with myocardial Infarction, most commonly in those who have significant left ventricular dysfunction.

In the present study 70% of arrhythmias occurred during the first hour of Hospitalization.

In the study by Aufderheide TP2, 90% of patients with acute myocardial infarction have some cardiac rhythm abnormality within 24 hours following infarct onset. In the present study 78.3% of patients with L.V. dysfunction had arrhythmias.

In a study by Ale jandro Macchia et al116 , compared to patients with EF >50%, systolic dysfunction patients had higher mortality and sudden death rates.

In a study by Yee Guan Yap et al108, in high risk post MI patients with LVEF < 40% or frequent VPCs, the risk of arrhythmic deaths was higher than that of non-arrhythmic deaths for upto 2 years. In a study by Scott D Solomon et al117, the risk of sudden death is highest in first 30 days after myocardial infarction among patients with left ventricular dysfunction, heart failure or both.

In the present study, majority of patients have arrhythmia during thrombolysis. The commonest arrhythmia during thrombolysis was VPC, followed by sinus bradycardia. In a study by Maria Cecilia Solimene et al118, reperfusion arrhythmias were observed in 75% patients and consisted of ventricular arrhythmias and / or sinus bradycardia. T

This study group was compared to another group with AMI treated conventionally and there was no difference between both groups in regard to the incidence and type of ventricular arrhythmia

In the present study, 65%% of patients consumed alcohol and 70%% of those who consumed alcohol had arrhythmia.

In a study by Djousse L et al119, it was concluded that there was little association between long-term moderate alcohol consumption and the risk of AF, but a significantly increased risk of AF among subjects consuming >36 g/day.

In the present study 55% of patients were smoker and 825% of those who smoke had arrhythmia.

In the present study, 40% % of patients were diabetics and 85% of those with diabetes had arrhythmia. In the study by Rana JS et al120, patients with

diabetes are less likely to develop ventricular arrhythmia than patients without diabetes.

In the present study 38% of patients were hypertensive and 80% of those with hypertension har arrhythmia .

CONCLUSION

In this study 70% of the patients had arrhythmia. Sinus bradycardia was the commonest arrhythmia. Ventricular premature contraction was a second most common arrhythmia. However ventricular premature contractions also occurred along with other arrhythmias like first degree heart block, sinus bradycardia, sinus tachycardia, right bundle branch block and ventricular tachycardia.

Arrhythmias were more common among elderly male .Arrhythmia commonly occur during the first hour of hospitalization.

SUMMARY

1. The current study in a hospital based descriptive study including 100 patients admitted with acute myocardial infarction.
2. Maximum arrhythmia were observed in the age group ranging from 50 to 60 years.
3. Arrhythmias were more common among elderly male.
4. Ventricular premature contraction was a second most common arrhythmia. However ventricular premature contractions also occurred along with other arrhythmias.
5. Majority of the arrhythmia occurred during the first hour of hospitalization.
6. Majority of the arrhythmias underwent spontaneous resolution.
7. Arrhythmias were noted more in those who underwent thrombolysis.

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ANNEXURE

PROFORMA

A PROSPECTIVE CLINICAL STUDY OF ARRHYTHMIAS IN ACUTE

MYOCARDIAL INFARCTION .

1. Name :

2. Age :

3. Sex : M/F

4. I.P. No. :

5. D.O.A:

6. D.O.D

7. Occupation

8. Monthly Income

9. Nature of work : Manual labour / office work / field work / work involving

important decision making / any other

10. Final Diagnosis

11. Complaint s Duration

a. Chest Pain

b. Dyspnoea

c. Palpitation

d. Light Headedness / Syncope

e. Nausea / vomiting

f. Sweating

g. Any other symptoms

12. Past History: Rheumatic fever/ diabetes / hypertension / ischaemic heart disease /

13. Family History : Hypertension / diabetes / I.H.D / Any other illness

14. Personal History : Diet veg / Non veg Sleep : disturbed / undisturbed

Habit s

Coffee / Tea. No. of Cups / day Alcohol Quant ity Durat ion Smoking Beedies / cigarettes No. per day

Paan with tobacco / without tobacco

15. Treatment History : History of any other medicinal drug intake
Name of the drug

Dose per day

16. PHYSICAL EXAMINATION

1. General Examination

Built and Nourishment : Well / moderate / poor

Pallor / Cyanosis / Jaundice / Clubbing / Edema / Lymphadenopathy
Xanthoma / Xanthelasma / Arcus Juvenilis / Arcus Senilis

2. Vital parameters

Pulse : Rate / min (Avg)

Regular / irregular

Regularly irregular / irregularly irregular
Character

Volume
Vessel wall

Radiofemoral delay
Apex pulse deficit

Blood Pressure mm Hg (Avg)

Respiratory Rate / min

Temperature

Jugular venous pulse.

3. Systemic examination
: C.V.S

InspectionPalpation : Apex
beat Percussion

Auscultation: First Heart Sound

Intensity : Norma l / abnormal / variable

If abnormal. Increased / decreased

Second Heart Sound

Intensity: Normal / Abnormal

If abnormal : increased / decreased

Splitting : Physiological / wide / reversed /
fixed Any murmurs

4. Respiratory system

Inspection

Palpation

Percussion

Auscultation

5. Abdomen

Inspection

Palpation

Percussion

Auscultation

6. C.N.S

Higher functions

Cranial nerves

Motor system

Sensory system

Reflexes

Signs of meningeal irritation

Signs of cerebellar dysfunction

Skull and spine

Carotid bruit.

INVESTIGATION

1. Urine analysis : Albumin : Sugar : Microscopy

2. Blood examination : Haemoglobin

Total Count

Differential Count

ESR :

Random Blood Sugar

Fasting Blood Sugar

Postprandial Sugar

Blood Urea

Serum Creatinine

Electrolytes : Sodium : mEq/L

Potassium : mEq/L

Chloride : mEq/L

Cardiac enzymes (at the time of admission to

ICCU, after 6 hours and 12 hours.

Lipid Profile

3. Chest X-Ray

4. Electrocardiogram

Time of Recording:

a. At the time of admission to ICCU

b. At 24 hours

c. At 48 hours

d. At the time of arrhythmia

Heart Rate : /min. Atrial rate : /min. Ventricular rate: /min

P. Wave

P.R. Interval Rhythm : sinus / nodal / atrial / ventricular /
dual Axis : Normal / right axis / left axis / indeterminate

QRS : Duration

Hypertrophy patterns : Atrial /
ventricular S.T. segment

QT duration
QTc.

Signs of ischemia / infarction
T. Waves

Arrhythmia :

a. How long is lasted

b. How was it terminated

c. Recurrence

5. Echocardiography

6. Any other investigation

7. Treatment given

8. Outcome and remarks: Arrhythmia: persisted / pharmacological intervention / electrical intervention / spontaneous resolution.

ABBREVIATIONS

ECG : Electrocardiogram

ICCU : Intensive Coronary Care

Unit CAD : Coronary Artery Disease

RBBB : Right Bundle Branch Block

LBBB : Left Bundle Branch Block

AMI : Acute Myocardial Infarction

SA node : Sino-atrial node

AV node : Atrioventricular node

MI : Myocardial Infarction

LCX : Left Circumflex Coronary Artery

LAD : Left Anterior Descending Coronary Artery

RCA : Right Coronary Artery

SVT : Supraventricular Tachycardia

QTc : Corrected QT interval

WPW syndrome: Wolff – Parkinson – White Syndrome

VPB / VPC : Ventricular Premature Beats / Ventricular Premature Contraction. VT : Ventricular Tachycardia

VF : Ventricular Fibrillation

t-PA : Tissue Plasminogen Activator

AF : Atrial Fibrillation

LAHB : Left Anterior Hemi-Block

IVCB : Intraventricular Conduction Block

CHB : Complete Heart Block

AWMI : Anterior Wall Myocardial Infarction

IWMI : Inferior Wall Myocardial Infarction

LVEF : Left Ventricular Ejection Fraction

L.V Dysfunction: Left Ventricular Dysfunction

IWMI+RV : Inferior and Right Ventricular Myocardial Infarction

ILMI : Inferolateral Myocardial Infarction

LWMI : Lateral Wall Myocardial Infarction

LVF : Left Ventricular Failure

CKMB : Creatinine Kinase – MB Fraction

CPK : Creatinine Phosphokinase

LDH : Lactate Dehydrogenase

ESR : Erythrocyte Sedimentation Rate

SGOT : Serum Glutamic Oxaloacetic Transaminas.

KEY TO MASTER CHART

Sl. No. : Serial Number

I.P. No. : In-patient Number

Htn : Hypertension

DM : Diabetes Mellitus

Smok : Smoking

Alc : Alcohol Consumption

Thromb : Thrombolysis

Time of arr : Time of arrhythmia detect ion

Arryth : Arrythmia

Arr.d.thromb : Arrythmia during thrombolysis

LVEF : Left ventricular ejection fraction

AWMI : Anterior Wall Myocardial Infarct ion

IWMI : Inferior Wall Myocardial Infarct ion

IWMI+RV : Inferior and Right Ventricular Myocardial Infarction ILMI : Inferolateral Myocardial Infarct ion

LWMI : Lateral Wall Myocardial Infarct ion

VPC : Ventricular Premature Contraction

LAHB : Left Anterior Hemiblock

SB : Sinus bradycardia

1s t deg HB : First degree heart block 2n d Deg HB : Second degree heart block

CHB : Complete heart block

ST : Sinus tachycardia

AT : Atrial tachycardia

VT : Ventricular tachycardia.

VF : Ventricular fibrillation

RBBB : Right bundle branch block

AF : Atrial fibrillation.

NAME	AGE	SEX	ARRHYTHMIAOCCURANCE	SITE OFINFARCTION	HTN	DM	SMOK	AIC	THR	TIME OF ARRH	TERM NTN	LVEF	TYPE OF ARRY
subbaiya	32	M	Y	AW	N	N	N	Y	N	1 hr	s	< 40	SB
Panchavarnum	43	M	N	IW	Y	N	Y	N	Y	1 hr	s	> 40	VPC+ST
pitchaiammal	59	F	Y	IW	N	Y	N	N	N	1 hr	s	< 40	VPC+ST
somu	26	M	N	IW	N	N	Y	Y	N			< 40	NO
ramanathan	60	M	N	AW	N	Y	Y	Y	Y			< 40	NO
karuppayee	56	F	Y	IL	Y	N	N	N	N	1 hr	pha	< 40	VPC
mallaiya	49	M	Y	IL	N	Y	Y	Y	N	1--12hrs	s	< 40	VT
thirunavukarasu	61	M	Y	AW	Y	N	Y	Y	N	1 hr	ei	< 40	SB
arokiaraj	55	M	N	IW+RV	N	N	N	Y	Y			< 40	NO
muthu	64	M	Y	IL	N	Y	Y	Y	N	> 24	pe	< 40	CHB
pandiyammal	54	F	Y	IL	Y	N	N	N	Y	1 hr	s	< 40	VPC
boominathan	52	M	N	LW	N	N	Y	Y	N			< 40	NO
john peter	68	M	Y	AW	Y	Y	Y	N	Y	1--12hrs	s	> 40	2DHB
balasakthi	44	F	Y	AW	N	N	N	N	N	1 hr	pha	> 40	CHB
sanjay	52	F	N	AW	N	N	N	N	Y			< 40	NO
fathima	65	F	Y	AW	N	N	N	N	N	1--12hrs	pe	< 40	SB
mathiyalagi	50	F	Y	AW	Y	Y	N	N	N	1 hr	s	< 40	AF
logeshwari	52	F	N	AW	N	N	N	N	N			< 40	NO
jeyarani	54	F	Y	AW	N	Y	N	N	N	1--12hrs	s	< 40	LAH
rajan	44	M	N	AW	N	N	N	Y	Y			< 40	NO
vasantha	45	F	N	AW	Y	N	N	N	Y			< 40	NO
pitchaiyammal	62	F	N	IW	N	Y	N	N	Y			< 40	NO
gomathi	57	F	N	IW	N	Y	N	N	Y			> 40	NO
vellaiyammal	64	F	Y	IW	N	N	N	N	N	1--12hrs	pha	< 40	VPC
ponnathal	69	F	N	LW	Y	N	N	N	Y			> 40	NO
anjeela	69	F	Y	AW	N	N	N	N	Y	12--24hrs	ei	< 40	SB
pitchaiammal	54	F	Y	IW+RV	N	Y	N	N	N	1 hr	s	< 40	AT
marimuthu	78	M	N	IW+RV	N	N	Y	Y	Y			> 40	NO
agilan	42	M	Y	IW+RV	Y	N	Y	N	N	1 hr	pe	< 40	VPC+RBBB
omprakash	25	M	N	IW+RV	N	N	Y	Y	Y			< 40	NO
vishnu	58	M	Y	AW	N	N	Y	Y	Y	1--12hrs	ei	< 40	VPC
jehangirkhan	46	M	Y	AW	Y	N	Y	Y	Y			< 40	NO
paramesh	54	M	N	IW	Y	Y	N	Y	N			< 40	NO
subbu	27	M	Y	IW	N	N	Y	Y	Y	1 hr	s	> 40	VT
mariyammal	34	F	Y	IW	N	Y	N	N	Y	1--12hrs	pha	< 40	2DHB

anantham	58	M	N	AW	N	Y	Y	Y	N			< 40	NO
puspham	36	M	Y	AW	Y	N	Y	N	Y	1 hr	pe	> 40	SB
natarajan	63	M	Y	AW	N	N	N	Y	Y	1 hr	s	< 40	VPC
ponmani	50	M	Y	AW	N	Y	Y	Y	Y	1--12hrs	pha	> 40	ST
kutti	35	M	Y	AW	N	N	Y	Y	N	1 hr	s	< 40	SB
vellaisamy	67	M	Y	AW	N	N	N	Y	Y	1 hr	ei	< 40	VF
agalyan	51	M	Y	AW	Y	N	Y	N	N	12--24hrs	s	< 40	VPC
murugeshwari	46	F	N	IW+RV	N	Y	N	N	Y			> 40	NO
arunachalam	41	M	Y	IW+RV	N	N	Y	Y	Y	1 hr	s	< 40	VPC+RBBB
nagammal	53	M	Y	IW+RV	N	N	Y	N	N	1 hr	pha	< 40	VPC
jeyalekshmi	63	M	Y	AW	Y	Y	Y	Y	Y	1 hr	s	> 40	SB
sekar	47	M	Y	LW	N	N	N	Y	Y	1 hr	pha	< 40	CHB
arumugam	56	M	N	IW	Y	N	Y	Y	N			< 40	NO
sekhar	29	M	Y	IW	Y	Y	Y	Y	Y	1 hr	pe	> 40	VPC
muthuraj	40	M	N	IW	N	N	N	Y	N			< 40	NO
rani	39	F	N	AW	N	N	N	N	Y			< 40	NO
krishnan	38	M	Y	LW	N	N	Y	Y	N	1 hr	s	> 40	VPC+SB
selvam	56	M	N	IL	N	Y	Y	Y	Y			< 40	NO
Krishnan	59	M	Y	IL	Y	N	N	Y	Y	1 hr	s	> 40	SB
Selvam	65	M	Y	AW	N	Y	Y	Y	Y	1 hr	pe	< 40	VPC
Suryaprabha	65	F	Y	AW	N	N	N	N	Y	1--12hrs	s	> 40	SB
Murugan	71	M	Y	AW	N	N	Y	N	Y	1 hr	pha	< 40	LAH
sahul hameed	60	M	N	AW	Y	N	N	Y	Y			< 40	NO
priya	62	F	Y	IW	N	Y	N	N	N	1 hr	s	> 40	VPC+SB
chinnamani	57	F	Y	IW	N	N	N	N	N	1 hr	s	< 40	SB
Arumugham	74	M	N	IW	Y	Y	Y	Y	Y			> 40	NO
Ramar	53	M	N	AW	N	N	N	Y	Y			< 40	NO
Sithiga beevi	43	F	Y	AW	Y	Y	N	N	Y	1 hr	s	> 40	VT+ST
Sampath	56	M	Y	AW	N	N	Y	Y	Y	1 hr	s	< 40	CHB+FDHB
Chandran	68	M	Y	AW	Y	Y	Y	Y	N	1 hr	s	< 40	VPC
Balakrishnan	51	M	Y	AW	N	N	Y	Y	Y	1 hr	s	> 40	SB
Perumal	54	M	Y	AW	Y	N	N	Y	Y	1 hr	s	< 40	VPC+SB
Nallasamy	58	M	Y	AW	N	N	Y	Y	Y	1 hr	s	< 40	SB
Mydeen pitchai	56	M	Y	IW	Y	N	Y	Y	Y	1--12hrs	ei	> 40	VT
Asokan	50	M	Y	IW	N	Y	Y	Y	N	1 hr	pha	< 40	AF
pandi	53	M	Y	IW	Y	N	Y	Y	Y	1 hr	s	> 40	VF
kaliyammal	67	F	Y	AW	N	Y	N	N	Y	> 24	pha	< 40	VPC+ST
Mahadevan	52	M	Y	IW+RV	Y	N	N	Y	N	1 hr	s	< 40	SB
Neelamegakan	57	M	Y	IW+RV	N	Y	Y	Y	Y	1--12hrs	pe	< 40	SB
nallayee	51	F	N	IW+RV	Y	N	N	N	Y			< 40	NO

kannaiya	73	M	Y	IW+RV	Y	Y	Y	Y	Y	1--12hrs	ei	> 40	ST
Rajendran	62	M	Y	AW	N	Y	Y	Y	Y	1 hr	s	< 40	VPC
Murugesan	49	M	Y	AW	N	N	Y	Y	N	1 hr	s	> 40	AF
Rethinam	63	M	N	IW	N	N	Y	N	Y			< 40	NO
Raman	77	M	Y	IW	N	Y	N	Y	Y	1 hr	pha	< 40	2DHB
Vellaisamy	52	M	Y	IW	Y	Y	Y	Y	N	1 hr	s	< 40	CHB+FDHB
Nagappan	55	M	Y	AW	N	Y	Y	Y	Y	1 hr	ei	> 40	SB
Seenivasan	64	M	Y	IW+RV	Y	N	Y	Y	Y	1--12hrs	s	< 40	VPC
malayan	51	M	Y	IW	N	Y	N	Y	Y	1 hr	s	< 40	SB
Arumugham	56	M	Y	IW	Y	Y	Y	Y	N	1--12hrs	pe	> 40	2DHB
Saroja	55	F	Y	IW	N	N	N	N	Y	1 hr	s	< 40	2DHB
Chinnaya	66	M	Y	AW	N	Y	N	Y	Y	1 hr	ei	< 40	SB
Rajaram	44	M	N	AW	N	N	Y	Y	N			> 40	NO
Sekar	61	M	Y	IW	N	Y	Y	Y	Y	1 hr	s	> 40	AF
Selvam	59	M	Y	IW	Y	N	Y	Y	Y	1 hr	pha	< 40	VPC
chandran	53	M	Y	IW	Y	Y	N	Y	Y	1--12hrs	s	< 40	AT
Karrupu	58	M	Y	IW+RV	Y	N	N	Y	N	1 hr	pha	> 40	2DHB
Gandhi	66	M	Y	AW	N	N	Y	Y	Y	1 hr	s	< 40	VPC+SB
Periasamy	65	M	N	IW+RV	N	N	Y	Y	Y			< 40	NO
Suresh	59	M	Y	AW	Y	N	Y	Y	Y	1 hr	s	> 40	VT+ST
Vellaisamy	45	M	Y	IW	Y	Y	Y	Y	N	1--12hrs	s	< 40	VPC
Kamatchi	57	M	Y	IW	Y	Y	Y	Y	Y	1 hr	ei	< 40	SB
Anand	76	M	Y	IW	N	Y	Y	Y	Y	1 hr	s	> 40	CHB
Majith	57	M	Y	AW	Y	Y	N	Y	N	1 hr	pha	< 40	VT
Alagar	55	M	N	AW	Y	N	Y	N	Y			< 40	NO

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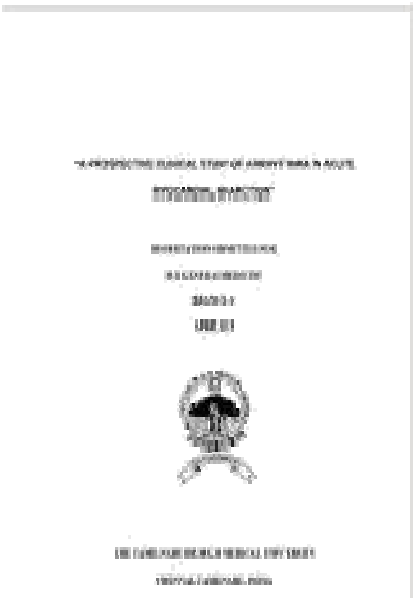

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BRANCH – I

APRIL 2015

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

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